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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

FACIAL PARALYSIS AND THE SURGICAL REPAIR OF THE FACIAL NERVE.*

DR. K. WINFIELD NEY, New York, N. Y.

Lesions involving the facial nerve are most commonly located at some point in its course through the facial canal in the temporal bone. Seldom are isolated lesions of the facial nerve observed within the skull. Intracranial lesions involving the facial nerve after its exit from the pons—usually tumors, meningitis, syphilis or aneurism—are rarely confined to the seventh nerve, and the associated involvement of neighboring nerves will assist in localizing the lesion as being within the cranial cavity. The strictly surgical lesions involving the seventh nerve within the cranial cavity are confined principally to pontine angles tumors, and do not fall within the scope of the present discussion.

Lesions of the facial nerve within the temporal bone are occasionally due to traumatism or otitic disease, but far more frequently the exact cause remains unknown. Facial paralysis is not uncommon in fractures of the base, the seventh being the nerve most frequently involved. The nerve may be completely torn at the line of fracture, or be impinged or crushed between fragments; compressed by hemorrhage within or without its sheath; stretched, or it may be only involved by shock.

Surgical trauma of the facial nerve during the radical mastoid operation is relatively infrequent in the hands of skilled operators,

*Read before the Otological Section of The New York Academy of Medicine, January 13, 1922.

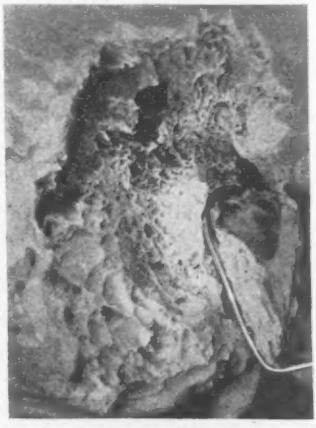
though some clinics report its occurrence in as many as 5 per cent of cases. In this operation the facial nerve is more frequently injured in breaking down the "bridge." If in opening the mastoid cells to reach the antrum, the evacuation is made too low and the penetration too deep, the facial canal may be opened and the nerve injured. The facial nerve is also especially liable to injury in curetting the posterior part of the tympanic cavity—sinus tympani—particularly if there is a defect in the wall of the facial canal.

Probably not over 7 per cent of peripheral facial palsies are found to be definitely associated with ear infections, and less than 1 per cent of all otitic suppurations are accompanied with facial nerve involvement. Facial paralysis is common in tubercular lesions of the ear, and though malignant disease of the middle ear is rare, it is usually associated with involvement of the nerve.

In 70 per cent or more of peripheral facial palsies, the etiology is still obscure and commonly ascribed to "chill" or "rheumatism," or considered to be an "infective neuritis." In many of these cases "exposure to cold" seems to be a definite factor. MacKenzie points out that many statistical cases have not had adequate ear examinations, and states that probably many of those cases of unknown origin would have been accounted for by a careful otological examination. The relation of the facial canal to the middle ear, and the frequency of bony defects in its wall, suggest a definite relationship between middle ear disease and facial paralysis, but the relative infrequency of nerve involvement in otitic infections, even of severe grade and prolonged duration indicates that the facial nerve is not particularly susceptible to otitic suppuration.

Our experience with a great number of infected war wounds has demonstrated that an intact nerve trunk is very resistant to surrounding suppurative processes, and that nerves may for many months lie in immediate contact with an active suppuration without functional impairment; but it has also been demonstrated, on the other hand, most emphatically, the susceptibility of nerves to compression, and the influence of apparently minor degrees of compression in the disturbance of their function. Certain anatomical peculiarities of the facial nerve (its course through a long bony canal which it entirely fills, and to which its sheath is intimately attached) are such as to convert even slight vascular changes into definite compressive factors, changes which in nerves not confined by a bony encasement would be productive of no disturbance, be-

cause of the elasticity of the surrounding tissue. Obviously, congestion, hemorrhage or the pressure of mild inflammatory exudates within or without the nerve sheath would be functionally destructive beyond all relation to their severity, because they are immediately converted into compressive factors by the unyielding character of the facial canal which the nerve normally fills. The firm attachment of the sheath of the nerve to the periosteum of the facial canal tends to localize these compressive agents rather than permit an adjustment by disseminating themselves along its course. A concomitant periostitis of the facial canal with an otitis media



1. (Photograph.) Dissection of temporal bone, showing course of facial canal in its vertical and tympanic portion—wire directed through canal. Posterior to the bend is the eminence of the lateral semi-circular canal; anterior to the nerve lies the tympanic cavity. Note the straight course of the nerve from its position anterior and below the eminence of the lateral semi-circular canal to the stylomastoid foramen. Identification of the nerve at these two points—just below the lateral semi-circular canal and at the stylomastoid foramen—is the keynote to the exposure of the nerve through its vertical course.

is probably the explanation of many of the facial palsies associated with otitic disease. Facial paralysis, in chronic otitis media with sequestration of the facial canal, is more apt to be due to pressure or stretching of the nerve, than to intraneural inflammation. Slight vascular or inflammatory changes may produce merely a temporary physiological interruption where the compression is only of sufficient degree to weaken or interrupt volitional impulses, which ultimately subsides, relieving the compression and permitting restoration of function. Compressive lesions of the facial nerve may develop to any degree, at times manifesting themselves merely by

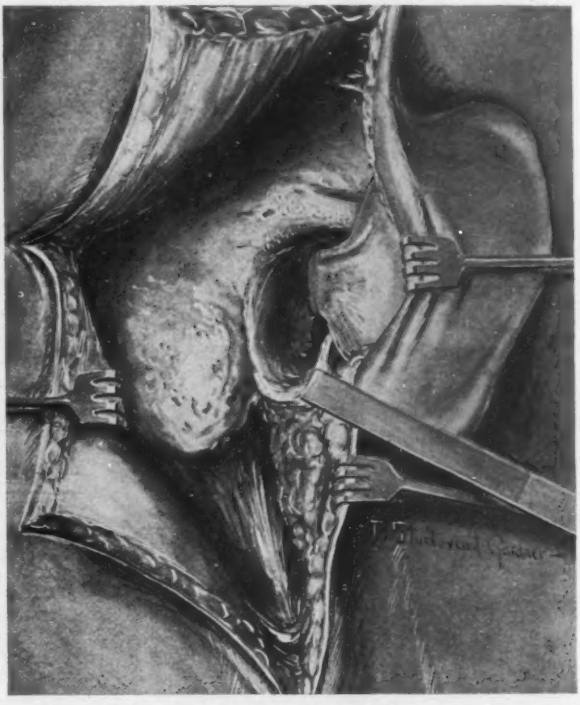
facial weakness or irritation (spasm), or they may be of such severe character as to produce complete degeneration of the neuraxons. If an acute lesion responsible for compression, such as hemorrhage or inflammation, is subsequently converted into scar tissue, the compression tends to remain more or less permanent.

The foregoing pathological conclusions have not been demonstrated in the facial nerve because of the difficulties of its surgical exploration in this region, and because such cases are seldom brought to autopsy, but the observation made on more accessible nerves in which it has been possible to study the nature and effects of different kinds of compression, lead us to believe that the factors considered above contribute largely toward the explanation of many facial palsies whose etiology is still unsolved. In the light of our present knowledge of peripheral nerve injuries and disease, the compression explanation seems to have a logical anatomical basis. Compressive lesions of the nerve trunk, demonstrated to be due to bone callus, constricting scar tissue about the nerve trunk, infiltration and thickening of the nerve sheath, or intraneural fibrosis following hemorrhage or infection producing constriction of the bundles, in yielding so successfully to surgical intervention, with such rapid restoration of function, following the removal of the compressing elements, proves conclusively the nature of compressive lesions and the results of their alleviation. Facial paralysis when associated with otitis media has been explained as being compressive in nature, due to pressure upon the facial canal through the accumulation of the products of intratympanic suppuration. I seriously doubt the possibility of an accumulation within the middle ear being sufficient to produce compression to a degree which would functionally affect the facial nerve even though it were exposed by a bony defect in the facial canal.

Occasionally, lesions involving the facial nerve distal to the stylomastoid foramen are produced by direct trauma, such as bullet or stab wounds, various surgical procedures, and in infants the application of obstetrical forceps. The facial nerve, as it passes through the parotid gland may be compressed by tumors, inflammatory products, scar tissue, or it may be subjected to surgical trauma. It is apparently very susceptible to the compression of malignant disease, and a parotid tumor associated with facial paralysis is strongly suggestive of malignancy.

The terminal branches of the facial nerve are frequently involved by accidental or surgical trauma, presenting certain difficulties to

the neuro-surgeon, because of the minute size of these branches, which do not readily lend themselves to surgical isolation and suture. These terminal lesions of the facial nerve which cannot be repaired by suture may leave distressing disabilities, though fortunately all the branches are rarely involved. It is for the correction of lesions of this type that muscle and fascia transplantation is at times justified.



2. Primary incision and exposure of the mastoid tip, suprameatal ridge and superior, posterior and inferior bony meatal walls. The chisel shows the point at which the removal of the auditory plate is begun, to permit exposure of the stylomastoid foramen.

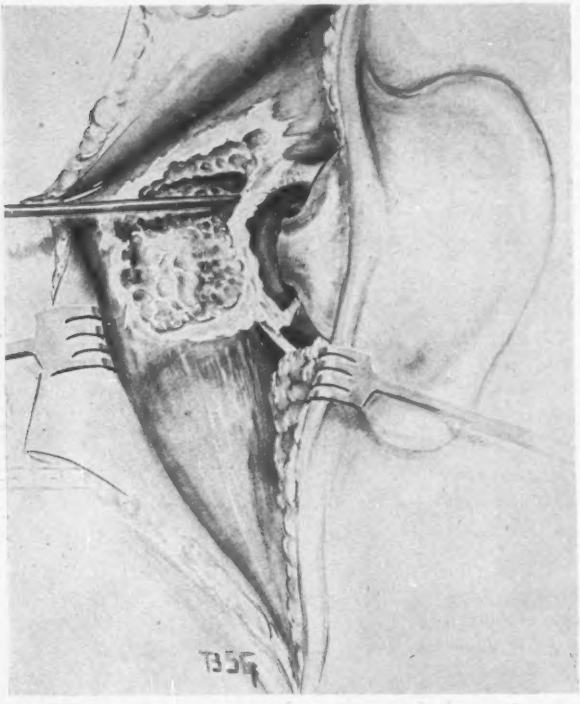
PROGNOSIS IN FACIAL PARALYSIS.

Most lesions involving the facial nerve within the temporal bone are probably compressive in nature. Even though the nerve be completely severed, its firm attachment to the periosteum of the facial canal does not permit its ends to retract, and they should maintain sufficient approximation to permit the regenerating nerve

fibers of the proximal segment to reach the distal segment. Failure of the facial nerve to regenerate following its division within the facial canal is likely due to the interposition of bone fragments, or its compression by the same. In most mixed nerves, there are usually sufficient cutaneous sensory fibers within the nerve trunk to determine neuraxon regeneration by Tinel's sign, but in the facial nerve we have not this assistance and must rely almost entirely for our information regarding its regenerative activity upon the reactions elicited in the facial muscles and upon the influence of time. In lesions of the facial nerve, as in lesions of all peripheral nerves, the determination of successful regeneration and restitution of motor function rests upon factors which can seldom be determined by a single examination, particularly when the lesion is of less than three months duration.

When the neuraxons of the distal segment of the facial nerve have undergone complete degeneration, following nerve division or progressive strangulation, the paralyzed facial muscles gradually lose their power to react to electrical or mechanical stimuli. These degenerative phenomena persist until the neuraxons have regenerated and again attained their neuromuscular junction, after which the electrical and mechanical irritability of the facial muscles again becomes evident and increasingly more active until their normal irritability has been regained. With the restoration of the neuromuscular end organ, the first visible manifestation of regeneration is the return of muscle tone, which when completely restored, leaves the face (when in repose) without deformity. Saliva no longer dribbles from the corner of the mouth, and tears cease to flood the cheek, the disability being evident only in attempting voluntary movement, or in emotional reaction. In the absence of neuraxon regeneration, the muscles may undergo a degenerative fibrous transformation which occasionally produces very deforming contractures. The first manifestation of fibrous contracture may be mistaken for a return of muscle tone, in that the contracture partially corrects the drooping angle of the mouth and the eyelid. These fibrous contractures may be differentiated from true muscle tone by the absence of electrical and mechanical irritability. In physiological interruption, where the compression has not been sufficient to produce strangulation of the nerve trunk with degeneration of the axis cylinders, the galvanic and myotatic reaction may remain active, though volitional movement fails to return. The preservation of faradic irritability is a favorable prognostic sign, and recovery may be expected within two or three months. When facial paralysis

continues for three months, with persisting modified electrical and mechanical reactions, the prognosis is less favorable. If the facial nerve has been completely severed within the facial canal, the regenerating axis cylinders, if regeneration has not been retarded, should reach the facial muscles about the fifth month, when muscle tone is regained; the sixth month should usher in the first evidence



3. Auditory portion of the tympanic bone, partially removed, exposing the exit of the facial nerve from the stylomastoid foramen to where it passes into the substance of the parotid gland. Mastoid cortex removed and cells evacuated—probe passed into antrum; suprameatal ridge partly removed.

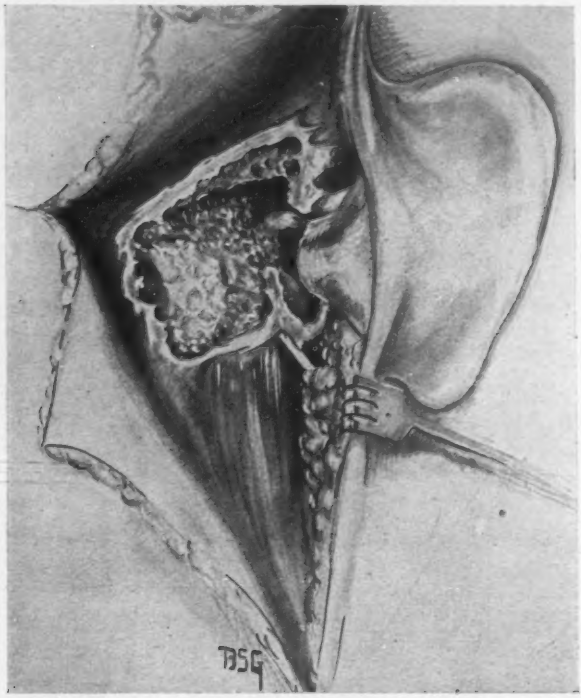
of returning voluntary power. A facial paralysis which shows no improvement in its electrical and mechanical reactions at the end of six months offers a poor prognosis, and should this condition continue unaltered for nine months, the possibility of spontaneous regeneration is indeed very small. After one year, it is practically hopeless.

NERVE ANASTOMOSIS FOR FACIAL PARALYSIS AND ITS RESULTS.

Little was done in a surgical way for the cure of facial paralysis until 1898, when Faure anastomosed the spinal accessory nerve to the facial at the suggestion of Furet. This procedure was introduced into England by Kennedy in 1899, and later Ballance proposed and carried out anastomosis with the hypoglossal nerve. After this, various operators attempted the correction of facial paralysis by anastomosis of the hypoglossal or spinal accessory with the facial nerve. Cushing, in 1902 did the first facio-accessory anastomosis in America as an original procedure, he at that time not being aware of the work of Faure and Kennedy.

The operation of nerve anastomosis, has been far from satisfactory, its physiological basis resting alone upon certain experimental work which had demonstrated the possibility of motor nerve fibers of a divided nerve to regenerate down the trunk of another nerve to which it had been anastomosed, and their ability to carry motor impulses. From an experimental standpoint this is evident, but it fails to take into consideration the functional specialization of the different areas in the motor cortex. With facio-accessory anastomosis, there was a return of muscle tone and the flaccidity of the face disappeared more or less at the end of four months. Usually between the fifth and sixth month some motion was observed in the formerly paralyzed muscles—occurring when attempts were made to elevate the shoulder or turn the head—calling into play sterno-mastoid and trapezius action. Though it was possible to get a restoration of motion in the facial muscles, this motion proved eventually disappointing, in that the facial movements were inaugurated through shoulder action and were in no way coordinated with facial expression or emotion. When at rest the face was practically symmetrical, but no voluntary movements were possible except when attempts were made to elevate the shoulder. Ballance and Stewart reporting on one patient, nine months after operation states, "Facial movements were so easily elicited by slight shoulder movements that the patient had to carry her parasol in her right hand instead of her left; otherwise involuntary facial movements so easily occurred that awkward misunderstandings with strangers resulted." Ballance believed that the hypoglossal center, which is so closely related anatomically to the facial center in the cortex could more readily be reeducated to take upon itself the function of the facial center (anatomical relationship does not necessarily indicate physiological relation). This procedure was likewise unsatisfactory; the motor control of the facial muscles now resided

in the hypoglossal cortical center and the facial muscles now responded instead of the lingual, especially noticeable during mastication and deglutition. Nature does not seem to take kindly to transferring the function of one cortical center to another, and I know of no case in which emotional expression has been conveyed to the facial muscles by way of the spinal accessory or hypoglossal cortical centers. It has been suggested that there



4. Bridge formed by the poterior meatal wall broken down over antrum, exposing the eminence of the lateral semi-circular canal; suprameatal ridge not sufficiently broken down.

might be some hope of reeducating the hypoglossal center to facial coordination if the anastomosis be done in the very young, but this does not seem to be borne out by experience, nor would it offer much hope to those who are unfortunate enough to develop persisting facial paralysis after maturity. The most that can be said as to final results in the cure of facial paralysis by nerve anastomosis is that muscle tone is restored, but with it emotionally irrelevant

and often embarrassing non-coordinated facial movements are added to the formerly expressionless facial muscles. Our conclusions drawn from the study of functional results following nerve sutures during the past war have demonstrated the individuality of the various fibers within a nerve trunk, and the inevitable functional complexities arising from a disturbance of their arrangement.

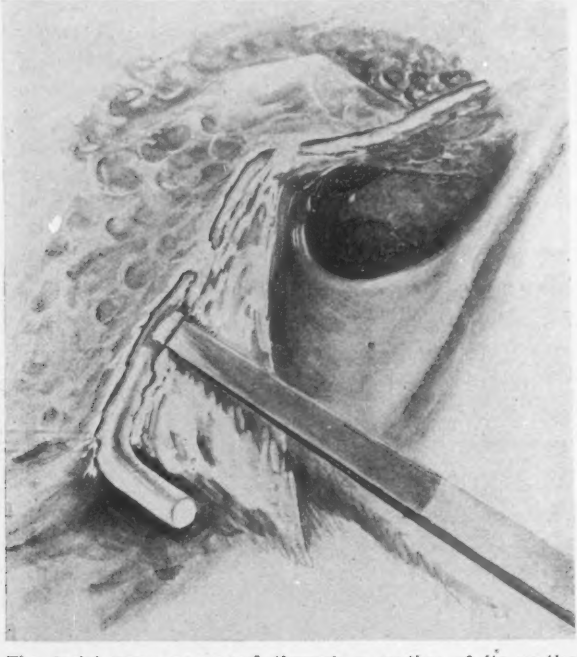
Obviously, the only hope of restoring bilaterally coordinated emotional expression after a paralysis of the facial nerve lies in the restoration of the functional integrity of that nerve. Surgical procedures devised with the hope of replacing the physiological activity of one nerve by another cannot hope, at least in one individual or in one generation, to change the functional characteristics of the controlling specialized cortical center. Hypoglossal fibers will continue functionally as hypoglossal fibers even though they be directed down the peripheral facial trunk. The anatomical misplacement of nerve fibers either by accident or design does not change their functional characteristics.

In literature, a few references are to be found relating to the direct repair of the facial nerve as it lies in the facial canal, but as a practical procedure—probably because of surgical difficulties—it has not been generally advocated. The realization that apparently our only hope for the correction of facial paralysis lies in the restoration of the functional continuity of the facial nerve, led me to attempt upon the cadaver various procedures directed toward the exposure of the nerve in its course through the facial canal. I found during this experimentation, that it was possible by careful dissection to liberate the facial nerve from the canal through the essential part of its course in the temporal bone without impairment of the vestibular or auditory end-organs. As a result of these investigations I became convinced that although the procedure was tedious, it is possible and practical to expose and attack the facial nerve at a point where its injury is most common, namely in its tympanic portion, where it passes between the fenestra ovalis and the lateral semicircular canal.

In those lesions of the facial nerve not due to injury, commonly called "Bell's palsy," and having the history of "exposure to cold," etc., the lesion is probably compressive in nature and located low in the vertical segment of the canal, in as much as the chorda tympani usually escapes involvement. In those cases which show chorda tympani involvement it is rare to find the lesion extending sufficiently high in the canal to produce a paralysis of the stapedius

muscle. These facts lead me to believe that in most cases of Bell's palsy the lesion is located within the vertical segment of the canal which may readily be exposed without endangering the contents of the middle ear.

Injuries of the facial nerve due to surgical trauma or fracture, involving the nerve in its tympanic course, are usually associated with a concomitant involvement of the air conducting apparatus, so that in the repair of these lesions it is doubtful whether the ex-



5. The facial nerve uncovered through a portion of its vertical and tympanic course, showing method of breaking down the wall with a fine, sharp chisel.

posure of the facial nerve in this region would materially increase the existing defect in air conduction.

ANATOMICAL CONSIDERATIONS.

At this point it would be well to consider briefly certain anatomical facts which have a direct bearing upon surgical procedures directed toward the exposure of the facial nerve. MacKenzie, in 1919, published a series of articles, the results of his anatomical study on "The Aqueduct of Fallopius and Facial Paralysis," from

which the following conclusions are drawn—most of which have been confirmed in my series of facial nerve dissections.

The facial canal may be conveniently divided for study into four segments, each of which will receive the following considerations: The *vertical* or *mastoid* segment of the canal runs from the bend or pyramidal segment almost vertically downward to the stylomastoid foramen, lying on an average of about 3 mm. posterior to the posterior meatal wall. In many instances the canal was separated from the mastoid cells by about 1 or 2 mm. of solid bone; in other specimens the cells were "adjacent" to the canal, being separated only by a thin shell of bone; while in still others the cells seemed to open directly into the facial canal. The *stylomastoid* foramen lies anterior to the tip of the mastoid, the distance averaging about 7 mm. depending upon the development of the mastoid process, the most "variable part of the mastoid bone." The stylomastoid foramen lies superior and internal to the mastoid tip, except in infants where the mastoid process is not developed. The *bend* or *pyramidal* segment is that portion of the canal where its direction is changing from the vertical to the horizontal. The bend is usually gradual, implicating from 2 to 6 mm. of the canal; though occasionally its direction changes with a sharp angle. The *tympanic* segment extends between the pyramidal segment and the genu; its average length being about 8 mm. About midway in its course, it comes into close relationship with the fenestra ovalis and stapes; it undergoes a gradual steady rise in level, reaching the junction of the roof and inner wall where it leaves the tympanic cavity, forming the genu. The *labyrinthine* section extends between the genu and the lamina cribrosa, averaging in length about 4 mm. This section though gently curved toward the cochlea, tunnels the dense petrous bone throughout its course, independent of the contour of the upper surface of the bone. The diameter of the facial canal is approximately the same at all ages, the labyrinthine portion being about 1 mm. wide; at the genu it broadens out from 2½ to 3 mm. and narrows again in the tympanic region to 1 or 1½ mm. The average length of the facial canal in the adult is about 25 mm.

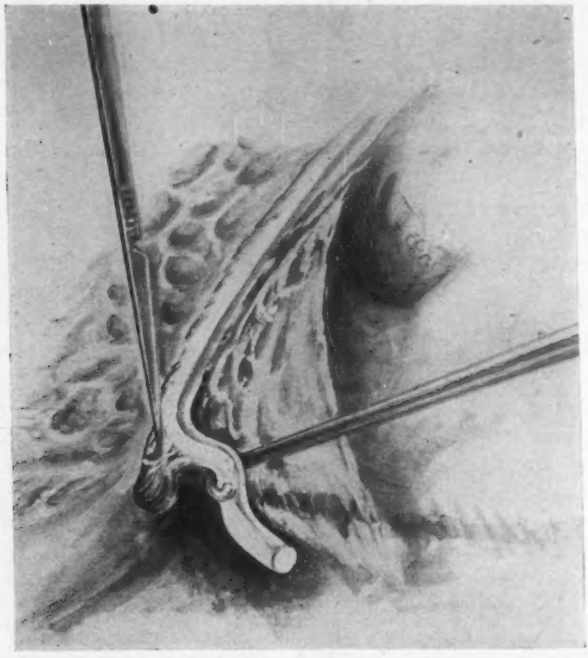
While there is considerable difference in the facial canal before the age of two years, after that date the dimensions, proportions and disposition are those of adult life, except in the length of the vertical segment, which is later lengthened through the development of the mastoid process. Up to the eighteenth month of life, the incomplete development of the mastoid process leaves the stylo-

mastoid foramen without bony protection, the facial nerve emerging from its canal on the surface of the skull.

TECHNIC FOR DIRECT REPAIR OF THE FACIAL NERVE.

The operative procedure for the exposure of the facial nerve in its course through the temporal bone from the genu of the facial canal to the stylomastoid foramen is conducted as follows:

With the patient under general anesthesia, the skin and subcutaneous tissues in the line of proposed incision, including the



6. The sheath of the facial nerve is firmly attached to the periosteum of its canal; its attachment being severed with a cataract knife, while the nerve is gently lifted from its bed. In this region the facial nerve is very friable and gentleness is necessary to prevent tearing the nerve trunk.

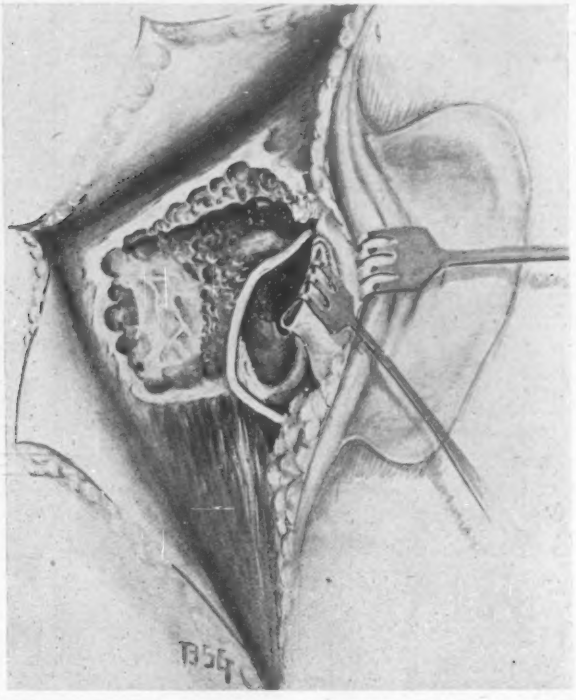
periosteum are injected with a 1 per cent novocain solution to which has been added 15 drops of a 1/1000 solution of adrenalin chloride to each 30 c.c. of novocain solution. The line of incision is similar to that commonly used in the radical mastoid operation except that it is extended forward above the ear to expose the zygomatic tubercle, and below one inch beyond the mastoid tip, following the

anterior border of the sternomastoid muscle. The infiltration is then carried to those structures lying immediately anterior to the mastoid process for the purpose of infiltrating the region of the stylomastoid foramen, constricting the stylomastoid artery. From behind the ear the needle is directed into the posterior wall of the external auditory meatus—hugging the bone—thoroughly infiltrating the entire superior, posterior and inferior meatal wall.

First Stage: The incision is now made in the soft parts and carried through to the bone. The soft parts are separated from the entire mastoid process, completely exposing the tip, care being used to avoid injuring the facial nerve in the region of the stylomastoid foramen. The separation of the soft parts is then carried anterior to the tip of the mastoid. The periosteum is now elevated from the supra-meatal ridge and carried as far forward as the zygomatic tubercle. The cartilaginous portion of the external auditory meatus is freed from its bony wall. When this separation is complete the cartilaginous portion of the meatus remains attached only to the anterior part of the tympanic ring, and the bony meatal wall is entirely exposed in its superior, posterior and inferior aspect. The attachment of the cartilaginous meatus to the auditory process of the tympanic bone is usually firm because of its roughened character—it is important that this plate be entirely exposed.

Second Stage: Before removing the mastoid cortex, I have found it advisable to remove the lower and posterior portion of the tympanic plate with a sharp chisel to a depth which will later give access to the stylomastoid foramen. After the auditory process has been removed, the operator may proceed to the removal of the mastoid cortex and the location of the antrum. The mastoid cells are now thoroughly removed, endeavoring to keep the cavity as shallow as possible by trimming down the rim left by their evacuation. This is particularly important in the super meatal region where the bony cortex and cells of the posterior zygoma should be completely removed as far forward as the zygomatic tubercle. The entire mastoid tip is now removed. The bridge formed by the posterior meatal wall over the antrum is broken down and the entire posterior meatal wall reduced to the level of the mastoid evacuation, remembering in this procedure the location of the facial nerve, using due care not to penetrate sufficiently deep to open the facial canal. The prominence of the lateral or external semicircular canal is now identified and immediately below and anterior to this structure, between it

and the tympanic cavity, lies the prominence of the facial canal. Occasionally the nerve will be found exposed, due to a defect in the canal wall, therefore great care should be used in this region, especially in breaking down the bridge. If, during the mastoid evacuation any suppurative process has been revealed, the exposure of the facial nerve should be postponed until the infection has been completely controlled.



7. The nerve removed from the facial canal throughout its vertical and tympanic course.

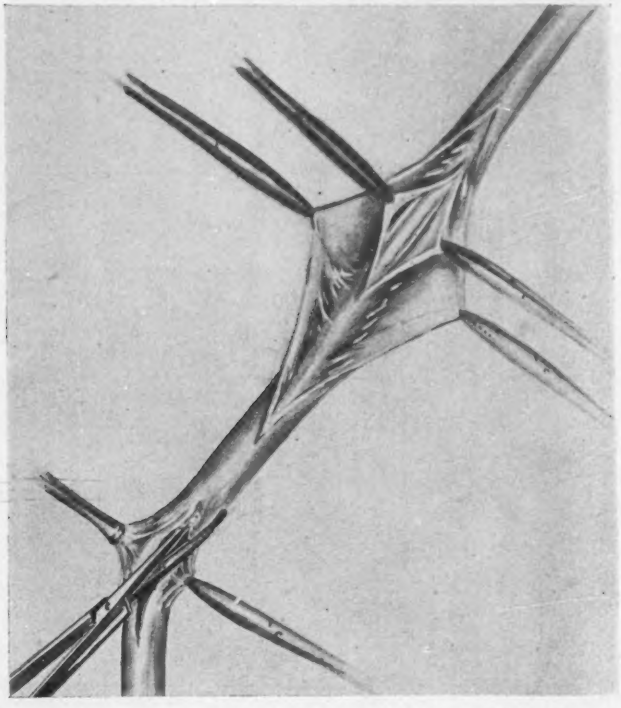
Third Stage: The facial nerve is now identified as it leaves the stylomastoid foramen, and the bone covering the vertical portion of the facial canal gently removed with a small sharp chisel, by careful scaling, until the nerve is exposed. If the operator bears in mind that the vertical portion of the facial canal follows an almost directly vertically downward course from its position below the lateral semi-circular canal to the stylomastoid foramen, he will be able to judge fairly accurately the depth of the canal and conduct

his evacuations accordingly. Often the vertical portion of the canal will present itself as a definite bony ridge, but occasionally the cellular structures open directly into the canal. After the nerve has been exposed throughout the vertical portion of the canal, the overhanging edges of the walls are carefully removed with a small sharp chisel. Small fragments of bone will often be found adhering firmly to the periosteum of the wall, which in turn is attached to the nerve. They should not be forcibly detached, but should be carefully dissected free with a cataract knife. The exposure of the nerve by gentle removal of the canal wall should now be continued through its tympanic portion as far as possible. This can usually be accomplished as far as the genu of the canal, providing the supra-meatal ridge and superior portion of the auditory meatus have been adequately removed.

Most surgical lesions involving the facial nerve are found to be in its tympanic portion near the lateral semicircular canal and if there has been a complete anatomical interruption of the nerve it should now be apparent. Compressive lesions, however, particularly if they are in the nature of internal compressions are seldom evident until the nerve is completely exposed and its sheath opened. In order to free the nerve from the canal, the periosteum, which is intimately attached to the nerve sheath, must be separated from its attachment to the bony wall. This is begun at the sides with a cataract knife, after which the lower portion is freed by gently lifting the nerve on a hook (beginning at the stylomastoid foramen) and carefully dividing its periosteal attachment to the canal. In this way the nerve may be freed throughout its course. During this procedure careful hemostasis must be observed; oozing points in the bone may be plugged with bone wax and blood clots washed away by a fine stream of saline solution. The facial nerve within the temporal bone is very friable and so easily torn that these manipulations must be conducted with great care.

Fifth Stage: The nature of the lesion will probably by this time be suggested by the appearance of the nerve. If the nerve has been completely divided, its ends should be sectioned and sutured, great care being given to the prevention of rotation or torsion of the nerve trunk—an accident which would ultimately greatly interfere with co-ordinated function. If the nerve is not divided, the lesion is probably compressive in nature, and most compressive lesions in peripheral nerves are not satisfactorily overcome unless the nerve sheath is opened, decompressing the nerve bundles. The following procedure should be instituted:

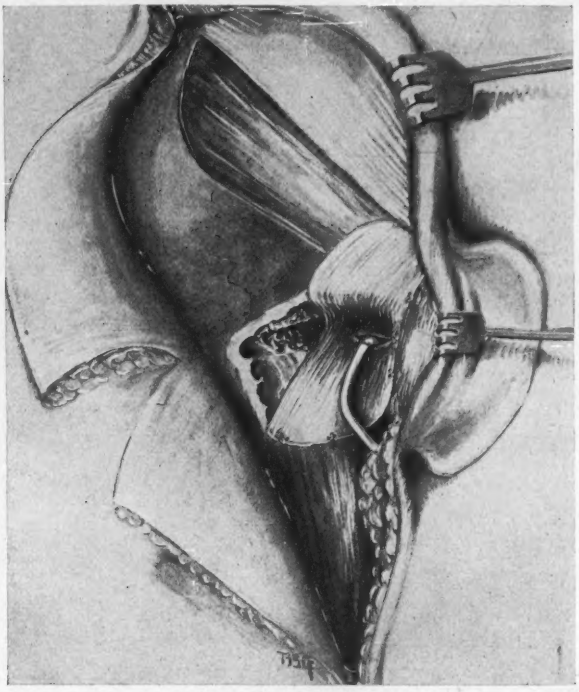
The nerve sheath is carefully grasped on each side with mosquito forceps and opened. When it has been entirely penetrated, the nerve structure appears as pearly white bundles which have a tendency to herniate through the opening. A pair of fine blunt pointed iris scissors are now inserted through the incision in the sheath, and the sheath divided throughout the length of the nerve. This



8. Decompression of the facial nerve by opening its sheath. Diagram shows the attachment of the sheath of the facial nerve to the periosteum of the canal, which has been preserved in the dissection. The sheath of the facial nerve is opened, exposing fibers which appear as a pearly white bundle.

procedure will usually suffice to completely decompress the nerve. At the point of compression the nerve bundles will often be found to be changed from a pearly white to a red or pink, with the nerve sheath adherent. If at the point of lesion the nerve bundles appear indurated so that the nerve trunk is hard to palpation, this portion should be resected and the nerve sutured. When it is

necessary to suture the nerve, if the defect be found too large, two procedures are open to the operator: (1) The position of the nerve may be changed and its course shortened by complete removal of the auditory portion of the tympanic plate, and of the posterior meatal wall permitting the nerve to take a direct course from the

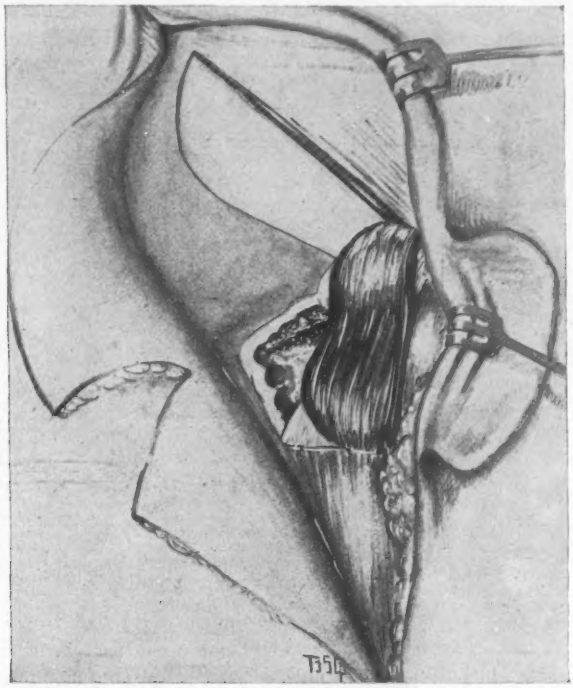


9. Plastic procedure to protect the nerve from subsequent compression by turning down a flap of temporal fascia which is passed under the nerve, separating it from immediate contact with the bone; method of anchoring the flap.

parotid gland toward the lateral semicircular canal. This will frequently correct a defect of about 1 cm.; (2) If this is not sufficient it will be necessary to resort to grafting, in which the sensory portion of the radial nerve in the forearm may be used, as it nearly approximates in size the facial.

If it should be necessary to immobilize the facial nerve throughout its entire course, it may be done at the expense of the auditory and vestibular end organs, by continuing the evacuation of the bone throughout its labyrinthine portion. If the nerve has been care-

fully uncovered throughout its tympanic portion to the genu of the facial canal, the operator will have the position of the nerve fairly well oriented, in that from this position its course changes to almost that of an acute angle, passing directly backward and inward to the lamina cribrosa. The depth of excavation from the surface of the lateral semicircular canal directly inward to the labyrinthine portion of the facial canal, averages about 9 mm. Although I have

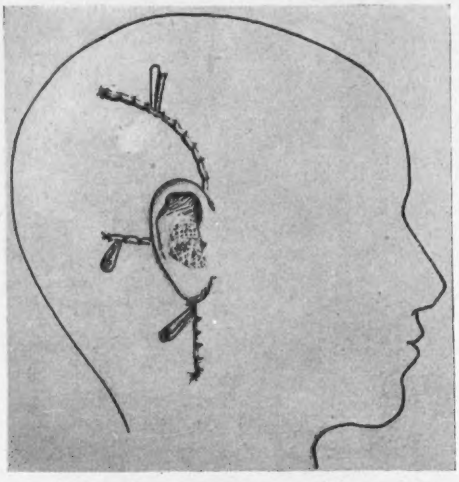


10. The portion of the temporal muscle denuded of its fascia, turned over the nerve. The facial nerve now lies between the flaps formed by the temporal fascia and the temporal muscle.

not yet had occasion to immobilize the nerve in this way as an operative procedure, I have repeatedly done so in fresh cadavers. Whether or not it will ever be justified at the expense of auditory and vestibular function is a question open to debate.

Sixth Stage: After the lesion in the facial nerve has been corrected, it should be protected from a subsequent compression. This is accomplished by one of two methods: The upper end of the incision is extended upward through the scalp, exposing the entire

posterior portion of the temporal fascia and its attachment to the temporal ridge. A flap of the temporal fascia is now carefully raised from the underlying temporal muscle, turned down into the bony excavation, slipped under the facial nerve and anchored. To more carefully protect the nerve and to permit of more accurate placing of this fascial flap, the squamous portion of the temporal bone just above the supra mastoid excavation may be partially removed, exposing the dura. After the nerve has been relieved of its immediate contact with the bone through most of its course,



11. Incision closed—points of drainage indicated. External auditory meatus lightly packed with iodoform gauze.

the cavity may be partially filled and the nerve further protected from scar compression by turning over it that portion of the temporal muscle which has been denuded of its fascia. In turning down this flap, the external portion of the muscle should be placed in contact with the nerve, as the temporal fascia may be separated from the muscle in this region with very little trauma, leaving a smooth, unbroken surface of muscle which is less apt to form adhesions than its under surface which suffers greater trauma in detaching it from the bone. The second method of protecting the nerve from its bony bed was found possible by lifting the temporal muscle and turning down a flap of pericranium, the upper surface of which should be placed in contact with the nerve.

After careful hemostasis, the wound is closed, two or three rubber tissue drains being inserted for any subsequent oozing (to

be removed at the end of twenty-four or forty-eight hours). The external auditory meatus is lightly packed with a strip of iodoform or B. I. P. gauze.

The subsequent treatment consists in daily massage of the face, beginning two weeks after the wound has entirely healed. Electrical treatment of the facial muscles, if successful in stimulating contractions, probably assists in improving their nutrition; it has, I think, no other value. A support to prevent the overstretching of the facial muscles made of adhesive as suggested by Yawger, or a hook inserted in the corner of the mouth as is commonly used, is of exceedingly great value, in that paralyzed muscles which are overstretched are slow to regain voluntary power.

CONCLUSIONS.

1. Peripheral facial paralysis is most often due to lesions which involve the nerve in its course through the facial canal in the temporal bone.

2. Most lesions within the facial canal are probably compressive in nature.

3. Facio-accessory and facio-hypoglossal nerve anastomoses are unsatisfactory procedures, in that all that may be hoped for is a return of tone in the facial muscles, but with the restoration of tone there is usually added incoordinated facial movements, irrelevant to expression or emotion, and frequently distressing—emotional expression not being restored.

4. The only hope of correcting persisting peripheral facial paralysis lies in the possibility of re-establishing the functional continuity of the facial nerve, thereby connecting the facial muscles with the facial cortical center.

5. It is possible and practical from a surgical standpoint to uncover the facial nerve in its course through the temporal bone, and to successfully institute suture or neurolysis, thereby correcting it anatomically and making possible a restoration of physiological continuity.

6. Non-traumatic facial palsies are usually located within the vertical segment of the facial canal and may therefore be decompressed without invading the canal in its tympanic segment, thereby preserving air conduction.

7. Lesions involving the tympanic segment of the canal are usually traumatic in nature, due to surgical accidents or fracture of the base, and are associated with impaired air conduction, which disability will not be greatly increased in most cases through exposure of the facial nerve.

34 East 64th Street.

THE LIVER TUNNEL AND CARDIO-SPASM.*

DR. HARRIS P. MOSHER, Boston.

Drawings by the writer.

Last year I reported the cadaver findings in some thirty adults in which the esophagus and stomach were injected with wax and sixty injected and dissected babies. I have recently supplemented these findings by certain clinical observations. A somewhat definite picture seems to be forming about happenings at the lower end of the esophagus.

The basic fact of my last year's paper was that the liver is chiefly responsible for the shape of the lower end of the esophagus. The lower end of the esophagus is usually cone-shaped or trumpet shaped, according to the closeness of the investing liver. The lower end of the esophagus has liver on the right, in front, and in many cases a thin tongue of liver hooks round its left edge like a sickle. Behind the esophagus is the descending aorta which separates it from the vertebral column. The subdiaphragmatic part of the esophagus runs, therefore, in a tunnel of liver.

During the past nine months in studying X-ray plates of my cases, in examining these cases before operation with the fluroscope. and in checking up the fluroscopic and plate findings with those obtained through the esophagoscope, the importance of the liver tunnel has steadily grown upon me. I have long been familiar with the fact that plates of the lower end of the esophagus often show it ending in a nipple like point. Between this and the fundus of the stomach there is a gap. This varies in length but may measure an inch or even an inch and a half. Finally I realized that this gap was the closed liver tunnel. This observation has proved to be a fundamental one, and has enabled me to connect up my anatomical and clinical findings.

It is this liver tunnel which I wish to speak of today.

THE LIVER TUNNEL SEEN THROUGH THE FLUROSCOPE.

The liver tunnel as studied on the cadaver, varies in length in different subjects. Its other dimensions also are subject to wide variations, and these variations are found at birth. The tunnel can be demonstrated by the X-ray plate or through the fluroscope. In

*Chairman's address. Read at Atlantic City, June, 1921, at the annual meeting of the American Laryngological Association.

watching a patient with a normal esophagus swallow, the esophagus is seen to come to a point momentarily at the upper border of the liver. Then after a delay of a second or two the liver tunnel opens up and the bismuth milk streams into the stomach.

I demonstrated in my last paper that the upper edge of the liver often makes a crease in the front wall of the esophagus, and that this crease is present at birth. The left crus makes a crease in the posterior wall of the esophagus and a marked notch in its left edge, so that there is often a balooning at the extreme lower end of the

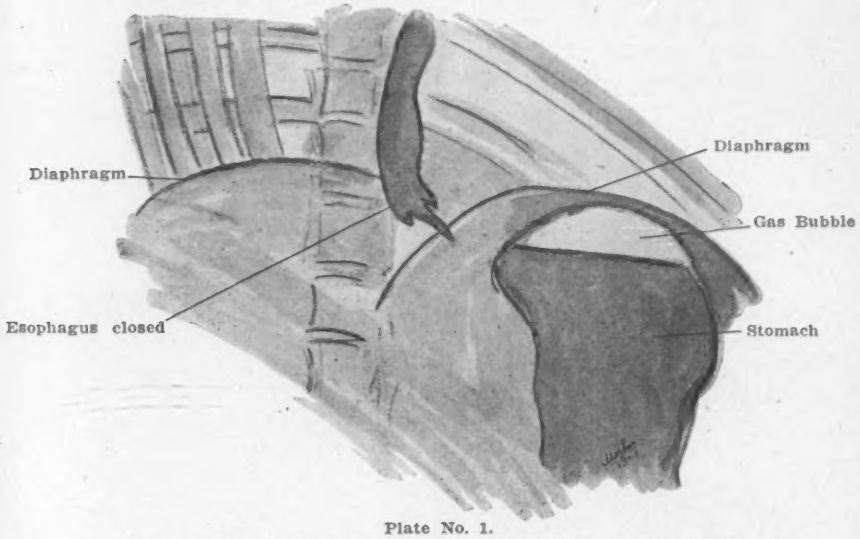


Plate No. 1.
Young male. Tracing of X-ray plate taken standing. The diaphragm is up in forced expiration. The esophagus is shut off and ends in a point.

esophagus just above the notch made by the left crus. The stage is set for a dilitation of the esophagus at this point. Above the crease in the front wall of the esophagus made by the upper edge of the liver a spindle shaped dilatation of the esophagus is common. This also is frequent even at birth.

Experiments on the cadaver show that the crescentic mound which is often seen through the esophagoscope in the right half of the field as the liver is approached is made by the upper rim of the liver. I asked Dr. Ross Golden of the X-ray Department of the Massachusetts General Hospital to prove for me that the upper

border of the liver exerted appreciable pressure on the front surface of the esophagus. I wanted if possible to duplicate the cadaver findings. The result of our combined observations is that in a majority of normal cases there is an appreciable delay in the barium milk at the upper border of the liver when a patient swallows.

Dr. Golden made the further observation that when the diaphragm is lowered the barium which is held back momentarily by the liver edge shoots at once into the stomach. Putting the patient

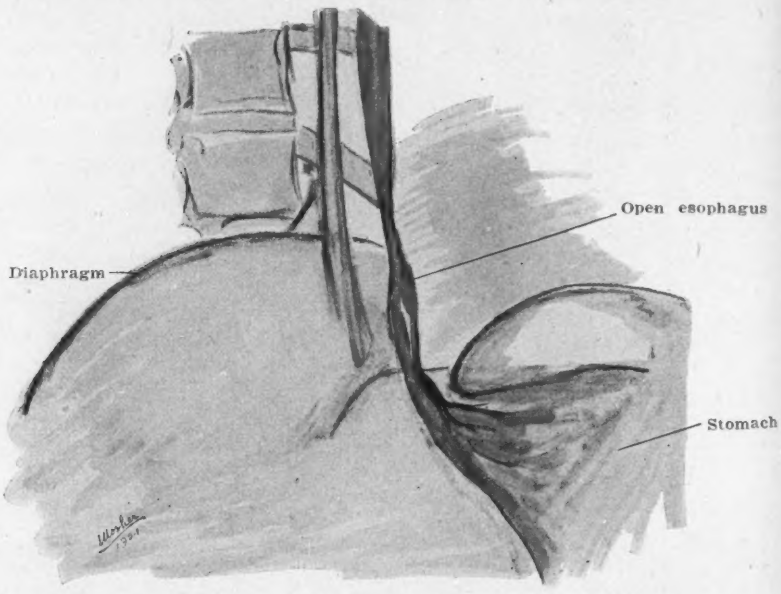


Plate No. 2.

Young male. The same patient as in plate No. 1. The diaphragm is down in forced inspiration. The esophagus is not constricted and the barium milk flows freely into the stomach. Plates Number 1 and 2 have deen duplicated in the majority of the normal cases so far examined.

in a position which causes the liver to fall away from the esophagus and so relieving the esophagus of liver pressure, also seems to make the fluid pass into the stomach more easily. This is probably one reason why we automatically sit up to drink.

Cadaver Experiments: Pulling the diaphragm down by traction on the left lobe of the liver tends to open the esophagus. Grasping the diaphragm near the hiatus and pulling downward also

tends to open the esophagus. If the left edge of the liver does not actually surround the esophagus but simply lies on its front face, backward pressure in such cases, cadaver observations show, causes an oblique line on the front face of the esophagus, the line corresponding with the upper edge of the liver, and the backward pressure of the liver tends to close the esophagus, the point of actual closure being on the left and below.

The Funnel of the Diaphragm: As the diaphragm dips down to form the hiatus or the esophageal opening, it forms a funnel. For an inch and a half above the hiatus the front and sides of the funnel are attached to the esophagus by connective tissue. Between the esophagus and the aorta behind there is the same connective tissue but the esophagus is less firmly bound down behind than in front and on the sides.

Annular Stricture Found in the Cadaver: I have found in the cadaver four specimens of annular stricture of the lower part of the esophagus. Each one caps a circumscribed dilitation of the esophagus which is bounded below by the constriction caused by the left crus. Each stricture is on a level with the upper edge of the left lobe of the liver. One specimen was dissected to show the nature of the stricture and it was found to be cicatricial. The lower part of the esophagus seems to be caught between the liver edge above, and the left crus below.

The Physiological Movements of the Diaphragm: The upward excursion of the diaphragm is caused by the relaxation of its muscular parts, by the rise of the intra-thoracic contents which are attached to the diaphragm, namely the lower lobes of the lungs, and the heart and its surrounding pericardium, and by the upward surge of the abdominal contents. The falling of the diaphragm is caused by the contraction of the muscles of the diaphragm aided by the weight of the liver which is attached to its under surface. Were the liver not supported by the viscera below it, and were these not held in place by taught abdominal muscles the diaphragm could never alone hold up the liver and act against its seven pound weight.

The diaphragm contracts sixteen to eighteen times a minute and waves of peristalsis run down the esophagus at frequent intervals during eating and at irregular intervals between meals in order to carry away the saliva. Nowhere else in the intestinal tract does a piece of gut—the esophagus is a piece of gut the same as the intestine itself—pass through an independent and regularly contracting opening as the esophagus does in passing

through the diaphragm. For this mechanism to act smoothly there must be some correlation or rhythm between the peristaltic movements of the esophagus and the relaxations and contractions of the diaphragm. Many patients with cardio-spasm as the fluroscope shows, can constrict and close the esophagus by forcing the diaphragm downward. As children many of us had panic when we attempted to swallow a pill and could not get it down until we took some water, which we knew went readily and so cured the panic. A nervous patient may have first had his attention called or rather focussed on the esophagus by some trivial mishap in swallowing, like swallowing the wrong way, swallowing a small bone, or, what is almost as uncomfortable, swallowing a bolus of air. In this way the unconscious rhythm between the peristalis of the esophagus and the diaphragm became upset. As a result the bolus of food would meet at times a closed diaphragm and the natural bagging of the esophagus which exists just above the right and left crus would be accentuated.

When a large esophagoscope is carried down the esaphagus of a cadaver a sizable pouch is found to the left above the left crus, and a smaller pouch on the right opposite the right crus. If the esophagus is not distended by inflation with air the walls of the esophagus invaginate upward into the tube and narrow its lumen to a slit or to a round central opening. A bolus of food which arrived at the hiatus when it happened to be closed might do the same thing.

Falling of the Diaphragm in Cardio-Spasm: In three cases of cardio-spasm of which I have good records the diaphragm was moderately lowered in one and markedly lowered in the other two. Dr. Merrill who made these observations for me and Dr. Holmes, both of the Massachusetts General Hospital X-ray Department, report that in the other cases of cardio-spasm which they have seen the diaphragm is usually lowered. In these three cases of mine the downward excursion was only a quarter of an inch whereas the upward excursion was normal, that is from an inch to an inch and a half. In two of these three cases forcible lowering of the diaphragm would constrict the esophagus and stop the flow of the barium milk. With the upward excursion of the diaphragm the flow through the esophagus into the stomach would be resumed. In quiet breathing there was no interruption of the flow.

It will be observed that this finding is just the reverse of the findings in the healthy esophagus. In the majority of the normal

cases investigated so far there is a momentary narrowing of the esophagus at the superior edge of the liver when the diaphragm is up and the barium milk hesitates there a moment. When the diaphragm goes down and carries the liver with it the esophagus opens and the barium milk shoots into the stomach. This corresponds with the cadaver finding that when the liver is pulled downward

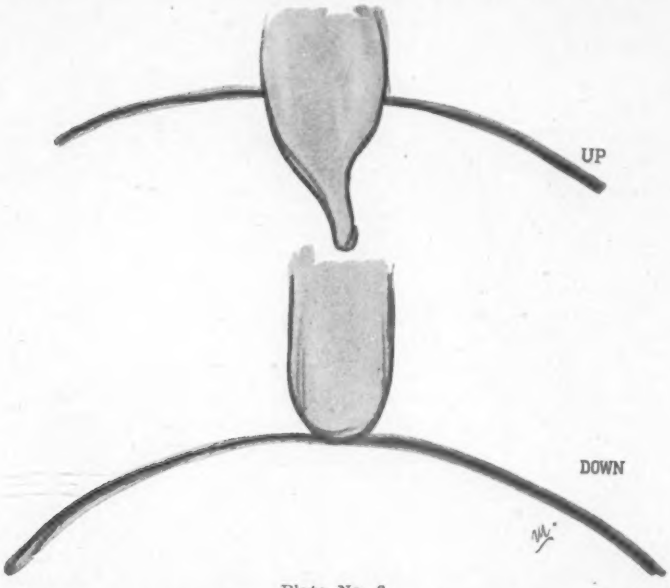


Plate No. 3.

Young female. Case of cardio-spasm. On examination with the esophagoscope a thin web-like stricture with a central opening—see plate No. 6, Fig. No. 1—was found at the level of the upper edge of the liver. In this case the diaphragm was slightly lower than normal. When the diaphragm was up in forced expiration the esophagus ended in a nipple like point and was partially open. When the diaphragm was down in forced inspiration the esophagus was shut off. In quiet breathing there was no interference with the flow of the barium. These two cuts were made from fluroscopic tracings some months after the web-like stricture had been divulsed. This finding is the reverse of the finding which so far has been found in the majority of normal cases. The percentages in which these two findings occur—the findings shown in plates 1 and 2 and plate 3—have not yet been worked out. So far most of the cases of cardio-spasm have shown a lowered diaphragm, but not all of them have shown the reversed finding of plate No. 3.

the esophagus tends to open. When the liver is up it exerts most pressure on the esophagus.

The Liver Tunnel and Cardio-Spasm: In grouping my cases of cardio-spasm I find that there is an element of stricture in the majority of them and this stricture is by preference at the beginning of

the liver tunnel, that is, at the upper edge of the liver. It has long seemed to me that it was asking too much of spasm to make it the sole cause of the extensive deformity which is present in long standing cases of cardio-spasm. To my mind it would be more rational to invoke some mechanical explanation either as the sole cause or at least as the continuing cause once the condition is initiated. The esophagus is a somewhat specialized portion of the general ali-

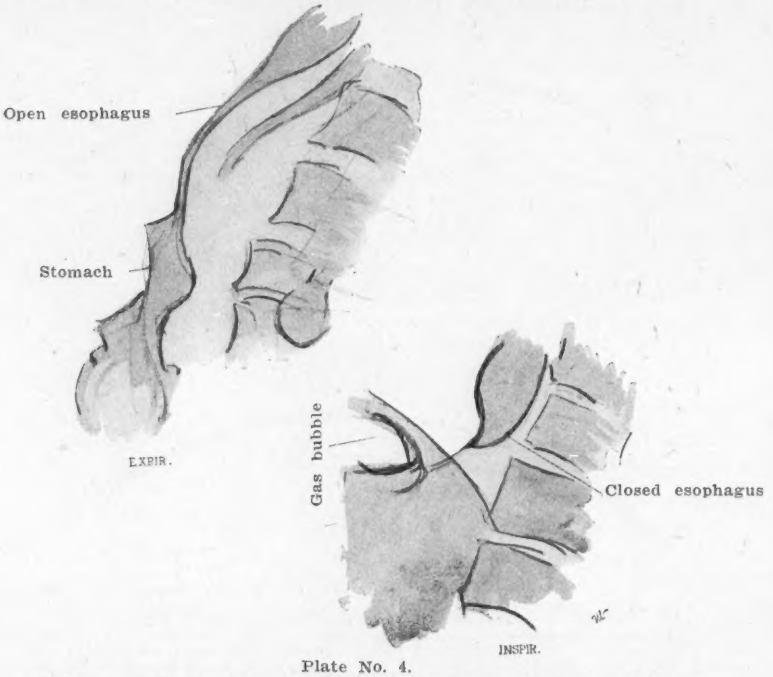


Plate No. 4.

Tracing of X-ray plate. (In the tracing right and left have been reversed through error.) This patient—sex, age and symptoms not recorded in my record—had a lowered diaphragm. In this case the reversed findings shown in plate No. 3 were present.

mentary tract, but like the intestines responds to irritation by contraction and spasm. I do not wish to do violence to physiology to the extent of ruling out spasm. I might be willing to allow it to start the vicious circle which ends in cardio-spasm. It is an annoying symptom of the disease in certain cases. It is surely capable of making bad matters worse. But why should it always occur at this one point in the esophagus? Why do patients with cardio-spasm seldom

have spasm or cramps anywhere else? I have met with but one patient who did. She was a young woman in the twenties who showed what the neurologists called a condition of tetany. Her nervous excitability remained that of a child. The threshold of all her nervous stimuli was very short. She was accustomed to have cramps in various parts of the body as well as the esophagus. I have watched for a duplicate of this case but have not found it. In my notes on this case there is the following record. She is one of the cases which I have been able to study most thoroughly owing to her willing co-operation.

Note—May 5, 1917. M. G. H. Fluroscopic examination, Dr. Merrill, two weeks after dilatation under ether.

For the last week the patient has eaten everything. With every meal there have been one or two spasms at the beginning of the meal. After these pass away there is no further trouble. By spasm the patient means halting of the food. She is conscious that the food halts and can tell the moment it begins to enter the stomach. When the patient has pain it is referred to the center of the sternum. She had pain but once and then only slightly last week.

On swallowing the barium milk it was seen to collect and to dilate the esophagus in the usual nipple-like manner. After two or three minutes and from this time on to the end of the examination, some ten minutes, the milk flowed steadily on in a stream of fully twice the size of the initial stream. This may have been due to the giving way of spasm or to the unfolding of the esophagus by the first food that passed down.

In quiet breathing inspiration and expiration had no influence on the rate of the flow of the barium through the esophagus into the stomach. On lowering the diaphragm by forced inspiration, the tapering point of the esophagus appeared as if suddenly cut off for an inch and a half, and the esophagus ended in a blunt point. On forced expiration the esophagus lengthened out an inch into its former pointed form. Forced contraction of the crura in this case closed the esophagus and forced relaxation opened it (see plate 3).

Note—The hiatus is an eliptical ring which surrounds the esophagus, obliquely crossing from left to right, and from above downward. The actual hole in the diaphragm through which the esophagus passes is tendious. The right crus is attached to the front of the esophagusan and the left crus is attached behind the esophagus to the posterior part of the margin of the hiatus. The hiatus being a tendinous hole, cannot close, but it narrows from below upward when the diaphragm ascends, and opens when the diaphragm descends. In certain cases of cardio spasm, as in the case just cited, the reverse happens. The reason for this is not yet clear.

Clinical Cases of Cardio-spasm Due to Stricture in the Liver Tunnel: In reviewing ten cases of cardio-spasm, I find there was a stricture in all. They are called cardio-spasm because the obstruction occurred at the lower end of the esophagus, the esophagus ended in the characteristic nipple-like point, and swallowing of cold fluids increased the difficulty, whereas swallowing of warm drinks made the passage of food through the esophagus easier. The stricture found in these cases varied from a slight crescentic fold in the right quad-

rant of the esophagoscope in the region of the hiatus to a full annular stricture with a central opening. In the cases which showed the crescentic fold steady pressure with the end of the esophagoscope usually resulted in the tube slipping by into the normal esophagus below and through this into the stomach. The withdrawal of the

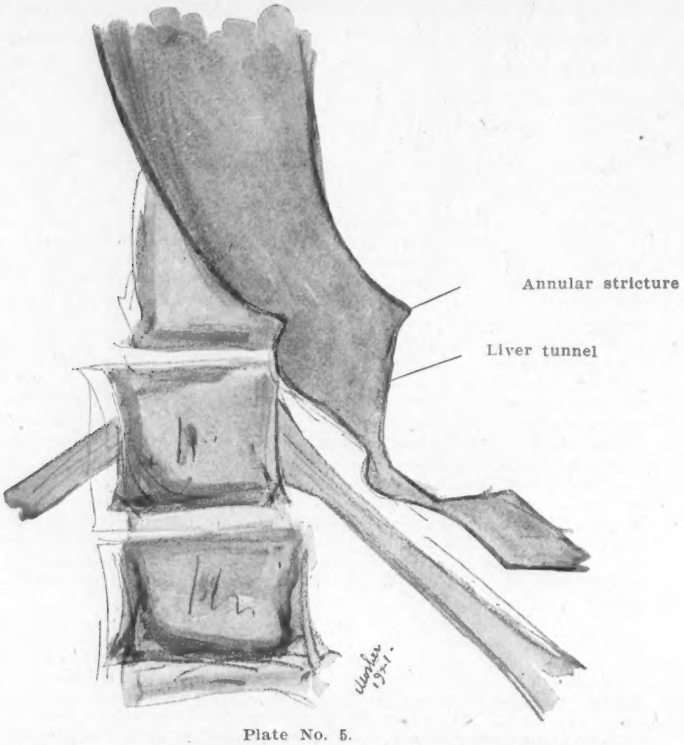


Plate No. 5.

Tracing of X-ray plate. Patient a woman of thirty-five. Typical symptoms of cardio-spasm. Some fifteen years ago the patient had the lower end of the esophagus dilated a number of times with a water bag. This gave marked relief. For the past two or three years the symptoms of obstruction have been returning. The plate shows an annular stricture of large calibre at the beginning of the liver tunnel. The lower end of the liver tunnel is closed and it is at this point that the present obstruction occurs. Examined under ether with the esophagoscope this narrowing was easily dilated . This patient left for the Far East much better of her symptoms. Probably sometime in the future she will need some sort of dilatation again.

tube disclosed a vertical slit in the mucous membrane of the esophagus. I have held these cases to be analagous to partial webs at the mouth of the esophagus. These act in the same way on the introduction of the tube.

The records show that three of the cases had a full stricture with a central opening. This stricture was at the hiatus or rather at the upper edge of the liver. On divulsing the stricture with the mechanical dilator a crescentic mounding was seen in the right field below it. Steady pressure caused the tube to pass this and enter the subdiaphragmatic esophagus and then continue on into the stomach. This mound I now believe to be the upper edge of the liver.

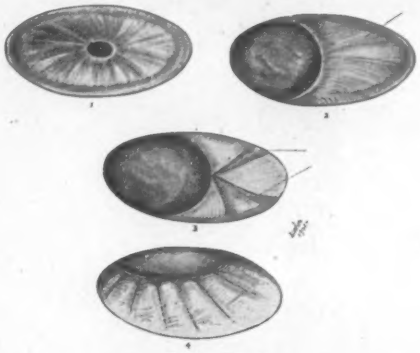


Plate No. 6.

Fig. No. 1. Web like stricture with central opening at the beginning of the liver tunnel found in four of ten cases of cardio-spasm. Fig. No. 2. Crescentic web-like stricture found in five of ten cases of cardio-spasm. This was placed at the beginning of the liver tunnel. On introducing the mechanical dilater, spreading it until marked resistance was felt and then withdrawing it closed, it was found that these strictures acted like webs at the upper end of the esophagus; that is, they were readily divulsed. On withdrawing the dilator one or two slits were usually found in the mucous membrane of the esophagus and the esophagoscope would then slip into the stomach without meeting obstruction. Below these web-like strictures the sub-diaphragmatic esophagus was normal. I feel that the upper edge of the liver is probably glued to the esophagus and reinforces these strictures. Fig. 3. This plate shows the slits in the esophageal mucous membrane after divulsion of the crescentic stricture. Fig. 4. This plate shows the normal sub-diaphragmatic esophagus usually found below the crescentic stricture.

Case in Which the Whole Liver Tunnel Was Narrowed: The following case showed a narrowing of the whole length of the liver tunnel. The esphagoscope demonstrated three partial strictures one succeeding the other at a lower depth and all just above the liver. At the edge of the liver there was a small central opening through which the tube could not be made to pass. The reason for this is seen at once by looking at the plate. This shows a narrow and long liver tunnel. It seems—to sum up the findings—that we can have strictures at the upper or lower end of the liver tunnel or anywhere in its course, or we can have a narrowed liver tunnel or a stiffened tunnel through which the food runs slowly. In any of these cases

as long as the esophagus retains its peristaltic action there may be an element of spasm from time to time which re-enforces the obstruction caused by the stricture or by the rigid or narrow liver tunnel. My observations would seem to show, however, that spasm is a minor element in these cases.

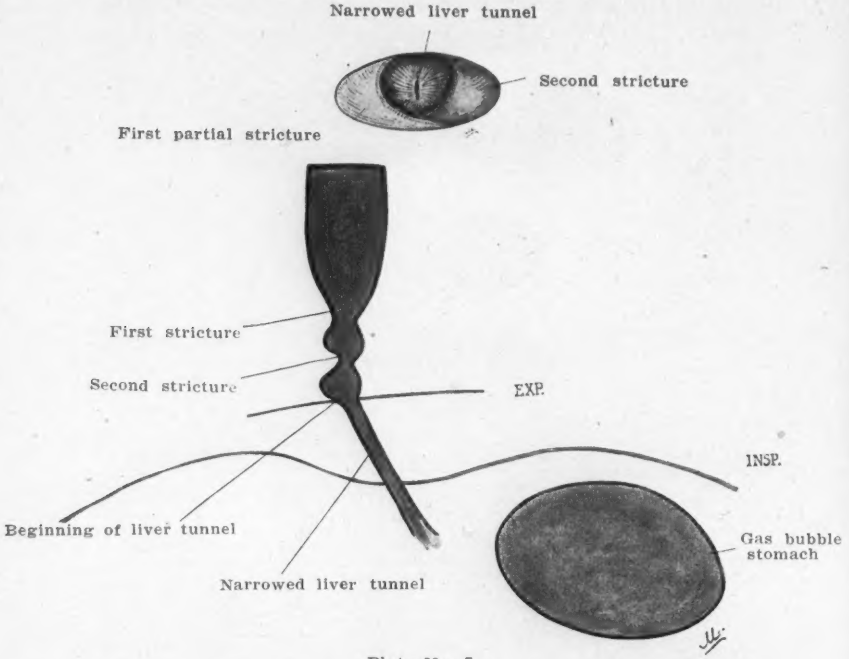


Plate No. 7.

Male, 45. Pre-operative diagnosis—cardio-spasm. Upper figure. The esophagoscope showed two crescentic strictures. The first was on the left and about an inch above the liver edge; the second was half an inch lower down and half an inch above the liver edge. The second partial stricture was on the right. The liver tunnel was narrowed to a central opening, which could be dilated to take only a No. 36 French elastic bougie. Lower figure. X-ray plate taken before operation. The plate shows the whole of the liver tunnel narrowed to a ribbon. The two strictures above the liver tunnel are shown in the plate to be really annular, not crescentic strictures, as they appeared through the esophagoscope.

What is the Cause of the Annular Stricture or of the Narrowing of the Liver Tunnel? The cause of the annular stricture at the upper edge of the liver tunnel or of the narrowing of the whole of the tunnel will be found, I believe, in some previous infection of the lesser omentum. The lesser omentum runs over the front face of the sub-diaphragmatic esophagus from the lesser curvature of

the stomach to the liver. Any abdominal infection which primarily or secondarily involves the lesser omentum, may result in temporary or permanent narrowing of the liver tunnel. Given an upper abdominal infection and infection of the lesser omentum, the constant hammering of the upper edge of the liver against the esophagus accentuates the involvement of the esophagus and determines a stricture opposite the liver edge. It is well known that temporary cardio-

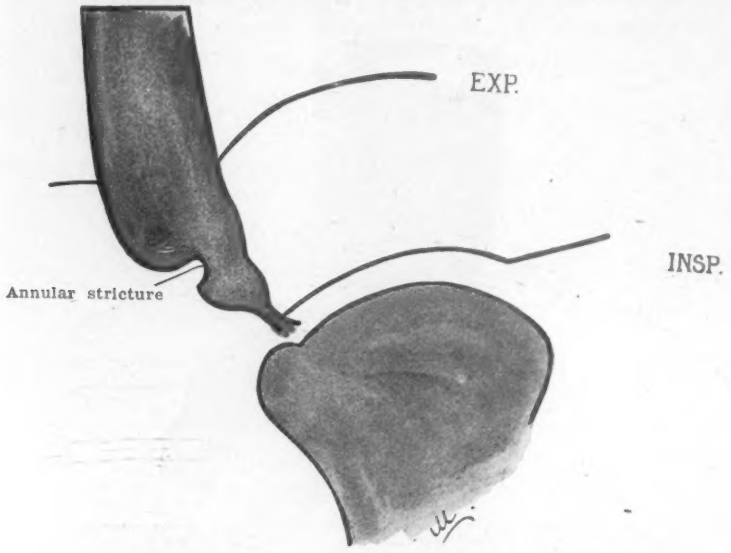


Plate No. 8.

Male, 50. Typical symptoms of cardio-spasm. Tracing of X-ray plate. Examination under ether with the esophagoscope showed a central web-like stricture. This was divulsed and below it in the sub-diaphragmatic esophagus an oblique band was seen running from right to left. Since divulsion the patient has been practically free from symptoms for four years. Plates taken recently show that the stricture is still present, yet it hinders the passage of food but little. Twenty years ago this patient suffered from an attack of general peritonitis of unknown origin. The plate shows a stricture about the middle of the liver tunnel. The lower end of the tunnel is narrowed. Compare with plate No. 5.

spasm is associated with disease of the gall bladder, cancer of the lesser curvature of the stomach, and with some cases of disease of the appendix.

In one of the cases of full stricture just recorded the patient when an infant swallowed a two-cent piece. On divulsing the central stricture the mound of the liver was seen, then the vertical folds of the subdiaphragmatic esophagus which terminated in an antro-pos-

terior fold or web. The esophagoscope would not pass this into the stomach. In another of these cases of central stricture the patient gave a history of an attack of general peritonitis twenty years before. The origin of this was never discovered. On divulsing the central stricture an oblique band was seen running upward and to the right. The tube would not pass this (see plate 8). Below the band the normal subdiaphragmatic esophagus was seen. The first case was probably traumatic, the second inflammatory.

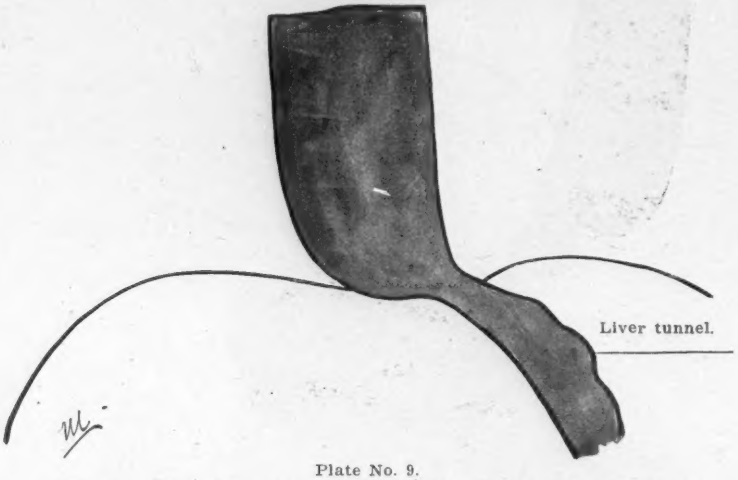


Plate No. 9.

Male, 35. This patient had a tubercular hip with an ilio-psoas abscess in childhood. There have been recurrent symptoms at intervals since. He had been examined under ether once before my examination. The examination was negative. My examination also was negative, as the large esophagoscope passed easily into the stomach. After ether, a No. 45 French elastic bougie could be passed readily. The patient's symptoms were relieved by the dilatation and the passage of the bougie only for a period of a few weeks, then the bougie would have to be passed again. The plate shows a ribbon-like liver tunnel which has lost its characteristic funnel shape. There is a slight constriction at the beginning of the liver tunnel. Above this the esophagus is dilated and sags to the right. The sub-diaphragmatic esophagus is out of line with the thoracic portion. The liver tunnel is narrowed probably from old adhesions. The passage of a bougie straightens out momentarily the two parts of the esophagus and stretches for a time the liver tunnel.

Cases of Involvement of the Esophagus by Extension of Neighboring Disease: After one period of dissection six bodies came to me and the esophagus and stomach were injected with wax in the usual manner. On dissection three of the bodies proved to be tubercular.

In the first tubercular glands encircled the esophagus from the clavicle downward. The glands were especially marked below

the bifurcation. The esophagus was so narrowed by the surrounding glands that the introduction of the large esophagoscope was impossible.

In the second body there was a tubercular infiltration on the inner surface of the middle and lower lobes of the right lung. The lung was glued to the pericardium and the pericardium to the esophagus. Adhesions ran from the pericardium to the esophagus. Opposite one of these there was a slight constriction of the esophagus.

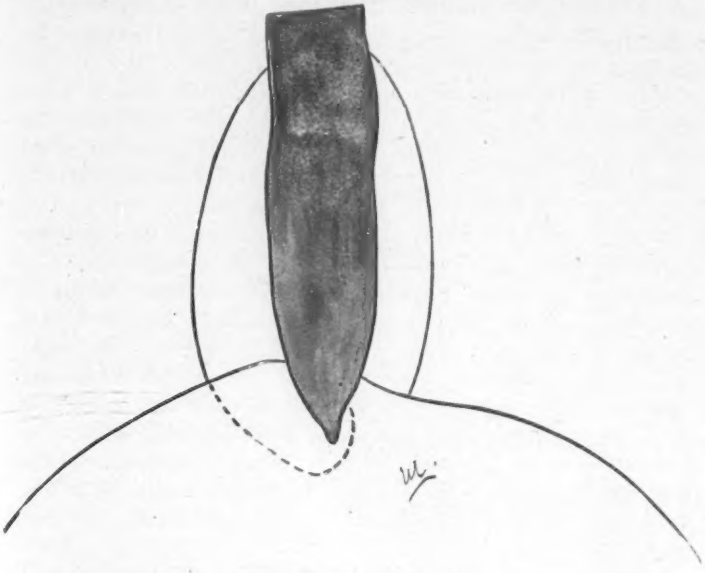


Plate No. 10.

X-ray tracing—right oblique position—of the same case as plate No. 9. There is a marked flattening of both leaves of the diaphragm in both plates. The esophagus is seen to come to a point in the center of the liver tunnel.

In the third there was a tubercular absecess in the lower lobe of the left lung. There were two tubercular nodules on the spine, one in the cervical region and one in the lower thoracic. Both nodules were healed. Opposite the lower one there was an annular stricture of the esophagus at the level of the upper edge of the liver.

These cases show that tuberculosis of the lungs and of the spine can effect esophagus. I wish to call especial attention to the fact that the annular stricture had the healed tubercular ulcer behind it, and that it was opposite the upper edge of the liver. My feel-

ing is that the pounding of the upper edge of the liver was the determining factor in the ulcerature of the esophagus which resulted in the stricture. There was no stricture in the cervical region opposite the site of the healed ulcer located there. In the cone of the diaphragm disease of the lungs or of the spine, therefore, may result in a stricture of the esophagus.

THE LEVEL OF THE POINT OF OCCLUSION IN CASES OF CARDIO-SPASM.

Where is the level of the nipple in which the esophagus ends in these cases of cardio-spasm? In the two cadavers examined in connection with this point it was found that the cardiac opening of the stomach was opposite the tip of the ensiform cartilage. With the falling of the diaphragm the liver, of course, falls with it and the level of the hiatus is correspondingly lowered. For instance, in one patient in which the esophagoscope demonstrated a stricture at the upper border of the liver the nipple of the esophagus was two inches below the tip of the ensiform cartilage and one inch to the left of the median line. The nipple was not at the cardia because the fluroscope showed the fundus to be an inch away. The esophagoscope showed a partial stricture at the upper border of the liver or at the entrance to the liver tunnel. The fluroscopic picture and the plate showed this partial stricture of the right half of the esophagus, below this the liver tunnel filled with bismuth. There was another stricture at the lower end of the liver cone. Here the esophagus showed the usual nipple-like point. The point of the nipple may therefore be at the top or the bottom of the liver cone or anywhere in between. In another case in which the diaphragm was low the nipple was in the middle line and one inch below the tip of the ensiform.

I have proved to my satisfaction that many so-called cases of cardio-spasm are mainly cases of stricture of some part of the liver tunnel.

828 Beacon St.

A NEW OPERATIVE TECHNIQUE FOR THE OPERATIVE AND POST-OPERATIVE TREATMENT OF MAXILLARY SINUS DISEASES.*

DR. H. B. LEMERE, Omaha, Neb.

There has been little change in the surgery of the antrum since the historic papers of Caldwell[1] and Luc[2], almost three decades ago. The various modifications of the operations described by Caldwell in 1893[1], all have the same principle underlying them, viz., after the pathological condition of the antrum has been surgically dealt with and the nasal opening obtained, that the oral opening should be promptly closed. It seems to me that some change in surgical technique is necessary to meet the requirements of the present understanding of the pathology of the antrum and of the importance of its low grade obscure infections causing general systemic infection manifesting itself in ear pathology, headaches, cardio vascular, nephritic and nervous conditions.

Often in these conditions the patients are very weak and nervous. Any local operation has so many terrors to them that they are quite unable to bring themselves to undertake it. In addition, any procedure would be dangerous which might transform an old latent infection into an active acute suppuration. It is to meet these two conditions that I have devised the present technique. The use of a general anesthetic removes the fear of pain and constant irrigation prevents active infection and absorption.

For several years I have felt that the early closing of the oral opening was undesirable because through it the antrum is much more accessible to post operative treatment and it is this modification of the operation which renders possible the surgical after-treatment, which I wish to emphasize. The following quotation is from Caldwell's original article:

"The frequency with which sinus disease is recognized is in proportion to the care with which the sinuses are explored.

"My own method in these cases has been to make a large temporary opening in the canine fossa through which the antrum is thoroughly explored, all deleterious material removed and the antrum thoroughly cleansed. A large counter opening is then made into the inferior

*From the Department of Otology, University of Nebraska, College of Medicine, Omaha, Neb.
Read before the Academy of Ophthalmology and Oto-Laryngology, Philadelphia, Oct., 1921.

meatus and *the primary opening closed. All subsequent irrigation drainage and medication is conducted through the opening in the inferior meatus.* By this method I have secured the best results with the minimum of inconvenience to the patient." (Italics mine—H. B. L.)

The following technique has the advantage which Caldwell's operation as originally given by him has of conservation of the greater part of the nasal wall and of the antrum mucous membrane. This is applicable to all latent and most empyema cases. It is not desirable in marked bony necrosis with sequestra.

Technique. The whole aim of the surgical treatment of antrum diseases as given in most works is drainage. While efficient and permanent drainage in a retention empyema produces such marked improvement that the case is considered cured, in most low grade infections and in many active infections much more than mere drainage is necessary to bring about a return to normal. Many cases show on operation two conditions: (1) a chronic diseased and thickened mucous membrane, (Fig. 1) and (2) either a marked softening or hardening of the nasal and the anterior walls (Fig. 2), especially noticeable in the thicker anterior wall. This change in the bone found on operating on the anterior and median walls undoubtedly extends to all walls of the antrum, but as the other walls are not interfered with in the operation their condition is not shown.

Many of these antrums are draining quite freely into the nose before any operation is performed and the natural openings seem quite patulous to the escape of secretions. The mucous membrane is pyogenic, however, and needs thorough treatment. It is for this reason that this operation and technique is devised, and that constant through and through washing is used for cleansing. Any method that provides thorough and constant cleansing will bring about resolution.

Ether anesthesia by the open method is used to commence narcosis. As soon as the operation commences it is necessary to have some form of ether blower and suction apparatus. I find the long suction point used in tonsil work very useful for the administration of the ether as it carries the ether well back into the mouth. A retractor is used on the upper lip (Fig. 4) and an incision about one inch long is made at the junction of the gingival and buccal mucous membrane, well above the roots of the biscusps and cuspid. (Figs. 3 and 4). The periosteum is elevated over the anterior wall,

(Figs. 3 and 4) and a circular opening made into the antrum, about three-fourths inch in diameter (Fig. 3). This is done either by Alexander mastoid chisels (Fig. 5), which allow the obtaining of a specimen of bone, or by drills. The Hudson hand drill (Fig. 6) provides a quick way for entering the antrum, the small burr being used first, followed by the larger burr. The location of the opening well above the roots of the teeth is shown in Figs. 3 and 5. It should be placed far enough laterally to escape the anterior dental branch of

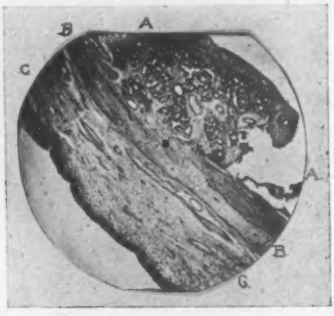


Fig. 1. Micro photograph of nasal wall from a case of latent masillary antrum disease, showing (a) greatly hypertrophied nasal mucus membrane, (b) bony nasal wall with invasion of fibrous tissue and (c) greatly hypertrophied mucus membrane of antrum.

Fig. 2. Micro photograph of anterior wall (canine fossa) from a case of latent antrum disease showing invasion of bone by fibrous tissue, a a'.

the infra orbital. A round mastoid spoon curette No. 3 is used to make the initial perforation through the nasal wall. First the posterior limit of the antrum is located by the curette, if the field is obscured by persistent bleeding this can be done by the sense of touch. The instrument is withdrawn slightly forward and the nasal wall broken through at its lowest point. A large size Ostrom forcep is now introduced and the blade engages the nasal wall through this opening. Still hugging the floor, the nasal wall is rapidly bitten

away under the inferior turbinate along the floor. The inferior turbinate thus escapes injury, it is indeed impossible to injure it if this technique is followed. I use a strong light and a head mirror and generally the operation is done by direct inspection, however, if it is impossible to keep the field free of hemorrhage, the work can all be done by the sense of location and knowledge of the anatomy, and the time shortened greatly. There is no attempt made to curette away the mucous membrane, except in a polypoid case,

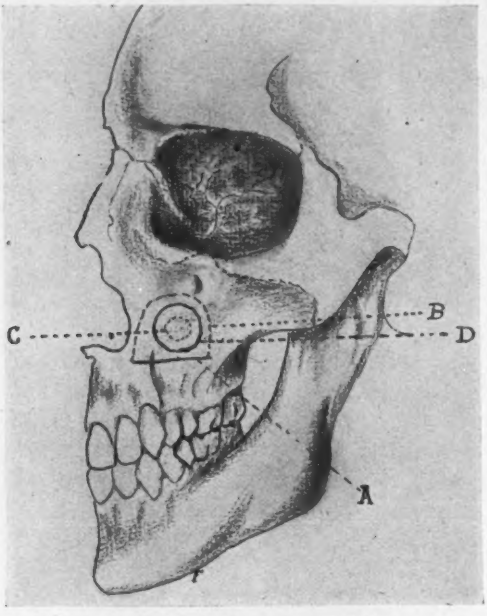


Fig. 3. Actual size. Skull showing position at (a) of incision into mucus membrane, (b) of elevation of periosteum, (c) of opening by small burr, ⅜ inch, (d) of enlargement of opening by large burr, ⅝ inch. A bony opening of this size is large enough to inspect the antrum cavity thoroughly if a head mirror is used, yet if properly placed it is small enough to escape injury to the infra orbital nerve, and to the roots and the nerve supply of the teeth.

when the curetting is done very gently. The object is always to preserve the mucous membrane. No astringents are used on the mucous membrane, such as strong zinc solutions, either at the operation or subsequently. They are found to be unnecessary.

The mucous membrane thus preserved and treated by the constant washing to be shortly described, will return so nearly to normal, that it makes a very much more desirable lining to the

antrum than the scar tissue resulting if the membrane is removed. This conservation of the antrum mucous membrane adds very greatly to the area of mucous membrane of the nose and its accessory cavities and to its function of moistening and warming the inspired air. The Ostrom forceps should be a large size, and strongly built. They are admirably suited to this operation as they cannot leave the inferior meatus nor injure the inferior turbinate, the inferior turbinate preventing the grasp of anything above its insertion. The operator thus feels every assurance that his opening is in the inferior meatus even if blood obscures the field. The

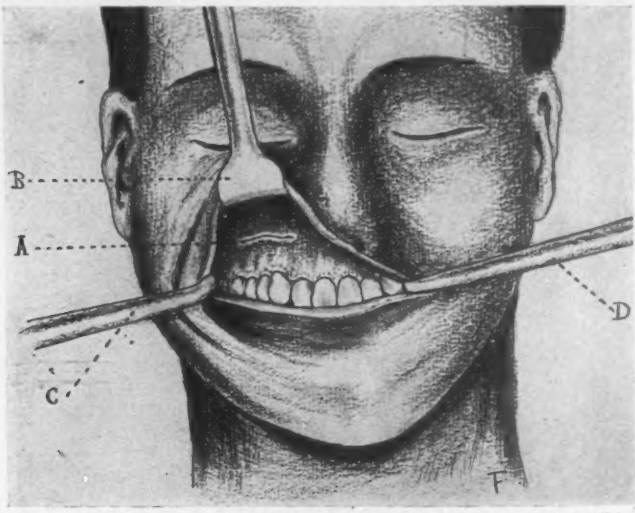


Fig. 4. (a) Incision into mucus membrane down to periosteum, (b) retractor, (c) suction tip, (d) anesthetic tip from ether blowing machine.

curette is now used as a probe to feel the size of the nasal opening obtained and if necessary to enlarge still further the anterior extremity of the nasal opening. One of the curettes should be somewhat heavier and stronger than the ordinary mastoid curette, as sometimes the nasal wall is of ivory hardness and if the curette head should break off while making the initial opening into the nose it might be very hard to recover it. If the opening into the nose is now felt to be amply sufficient, a rubber tube perforated in its upper portion is introduced into the antrum, pushed in as far as possible and the lower end allowed to project about three-fourths of an inch into the mouth. The incision is closed by one catgut

suture, central to the tube and the ends of this suture are tied several times around the tube and return to secure it in its position. (Fig. 7). The tube is not perforated by the suture as it is necessary later to wash through it. The tubes are now cut off short enough to be concealed by the lip. Care should be taken, however, that they are long enough to rest against the upper wall of the antrum or they may disappear into the antrum and be hard to recover the next morning. This completes the operation and the patient is returned to bed and the nurse instructed to keep him lying on his face until he is well out from his anesthesia. Actual

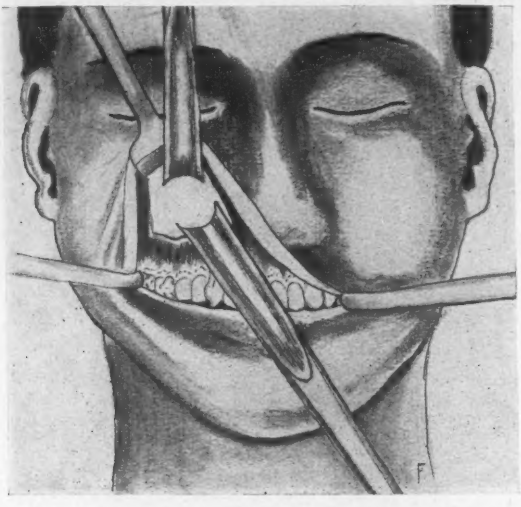


Fig. 5. Method of opening by Alexander chisels. This method is used in order to obtain a specimen of bone for microscope.

operating time is usually very short. The suction must be working well and should be applied to the pharynx and also within the antrum cavity itself. Extra suction tubes should be on hand in case one tube becomes obstructed either from bone dust or chips or from blood colts. A bowl of water to dip the suction end into aids materially in keeping the suction clear.

Washing. The washing out after operation is extremely simple. An ordinary soft rubber bulb ear syringe and a solution of one per cent biborate of soda solution is used. (Fig. 8). The syringe should be the red rubber in which the stem gradually increases in size rather than the gray rubber in which the stem is uniform up

to the bulb. The solution need not always be heated, it is better tepid, but can be used at room temperature. During the first twenty-four hours irrigations are commenced about six hours after operation, are carried on through the tube, and are used about every three hours. After twenty-four hours the tube is removed and irrigations are now carried on directly by the soft rubber bulb ear syringe through the oral opening every half hour during the day, and about three or four times during the night. The irrigations are very easily and painlessly performed. If a special nurse is not available, a relative or friend of the patient, or he himself

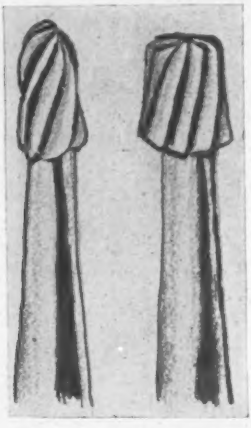


Fig. 6. Small (eccentric point, ⅝ inch) and large drill point ⅝ inch (actual size). Used with Hudson hand drill to make initial opening when no specimen is desired.

can be easily instructed in the procedure. The syringe should be in the patient's left hand for washing the left antrum, and in the right hand for washing the right antrum, as this gives the point the proper direction and it can be inserted without pain. The nose is blown after each washing, as sometimes the heavy secretion will otherwise remain in the nasal cavities. In practice it is found that the patients almost invariably learn to do their own washing on the second day and they are then allowed to leave the hospital on the third or fourth. They then return to their home and give their absolute attention to constant washing for about two weeks, returning to the office daily for observation. At the end of two weeks it becomes difficult to get the point of the syringe into the oral opening and the return flow by this time is generally clear. The

washing is continued through the gum as long as possible, but if the opening is patent and the return is clear at the end of two weeks the irrigations are lessened in frequency. A nasal wash is given to be used with a glass nasal douche after the oral opening has closed, this is used about three times a day. After the opening in the gum closes the usual nasal washing with a curved canula can be carried out by the nasal route as is done in the inter-nasal operation. This is seldom necessary as a routine for any length of time, but is used to determine if the antrum is remaining free from

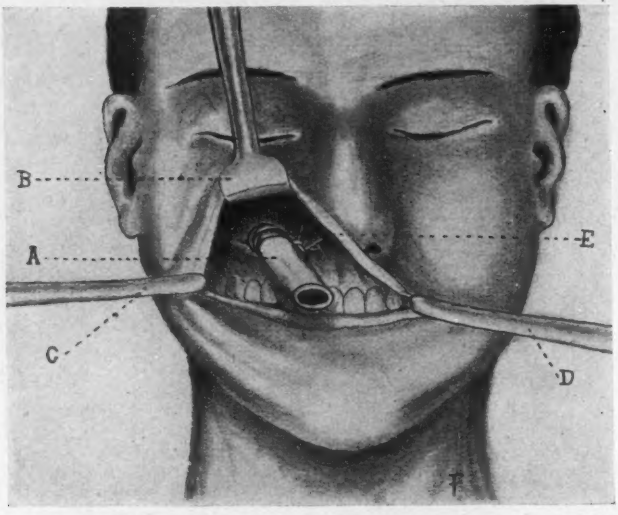


Fig. 7. (a) Rubber tube used for irrigating. Removed with suture in twenty-four hours, after which irrigation is carried out as in Fig. 8. (b) Retractor, (c) suction tube, (d) ether tube, (e) suture through mucus membrane, tied and passed several times around tube.

pus. Often, some little time after the antrum is apparently clear, an acute infection takes place which, however, subsides in a few days by daily washings of the antrum through the nasal opening. It is comparatively easy for most patients to, themselves, insert a Eustachian catheter through the nose into the antrum and then with the same rubber syringe they used for oral washing irrigate through the catheter. The tip of the rubber syringe fits perfectly into the end of the catheter without any other attachment.

Severity of Operation. The view in which oral operations are held by the rhinologists generally is well expressed by Dr. H. W.

Loeb[3], in his "Operative Surgery of the Nose, Throat and Ear." He states in regard to oral operations, including Caldwell's, Luc's, Denker's, Beck's and Partsches' operations:

"Very good results follow these operations as good drainage is established through the nose and reinfection is uncommon." He adds, however, "various accidents and other unpleasant results may follow these operations. Among them may be mentioned aspiration pneumonia, anasthesia of the cheek or teeth, emphysema

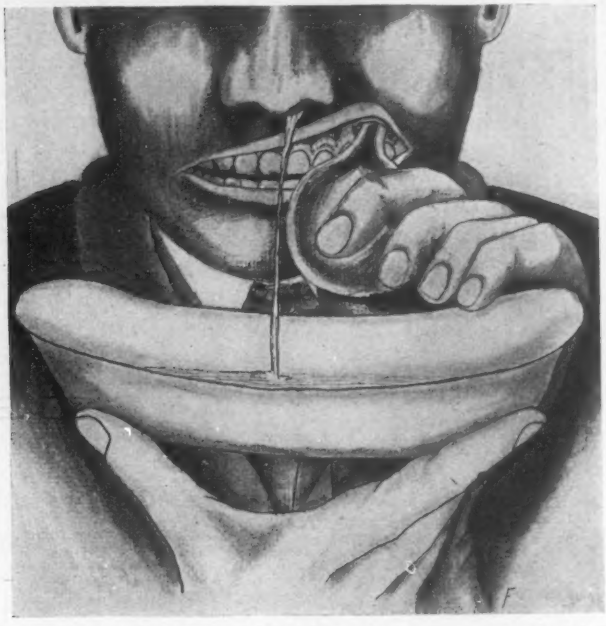


Fig. 8. Through and through washing carried out by the patient or nurse every half-hour during the day, and two or three times during the night, until the opening closes itself, which is usually in two weeks. The opening has never remained longer than three weeks. Irrigating fluid is Sod. bibor. (borax) .05 per cent.

of the cheek, neuralgia of the fifth nerve and osteomylitis of the maxilla."

Of these accidents and unpleasant results out of a series of over one hundred cases, I have had only the anesthesia of the lip occur and this has always in time cleared up.

There has been occasionally apprehension expressed that the nerve supply to the teeth may be injured. I have seen no evidence

of this though I have studied carefully X-rays of the teeth as long as two years after the operation. These showed absolutely no change in the apices, nor in the cancellous structure of the surrounding bone. On subsequent washings of the antrum months after the operation, the patient generally says he feels it in his teeth, showing the nerve supply is intact.

Appearance of the Nose after Operation. After healing, the examination of the nasal cavities usually shows a normal appearance, and there is no sign of any surgical interference. Sometimes the opening is visible beneath the inferior turbinate, as a rule, however, it is impossible to see it, though it can be readily found with the curved canula used for washing after the oral opening is closed. There is an absence of the scabs or mucopurulent secretion seen

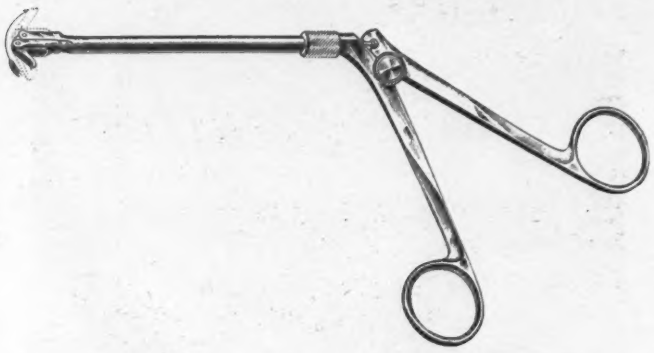


Fig. 9. The Ostrom cutting forceps, used in cutting out the nasal wall. The primary opening is made by a bone curette at the posterior inferior portion of the nasoantra lwall. The Ostrom forcep then bites out all the bony wall of the inferior meatus, especially enlarging the anterior end of the opening.

in the middle fossa before the operation. In cases which have shown atrophy before operation, the turbinates show a marked tendency to re-filling and the nasal cavity returns much nearer normal than would be thought possible. When suffering from a cold the infected secretion from the antra can often be seen coming from under the inferior turbinates, but the patient's report is that colds are less frequent, are never severe and always clear up after a few nasal washings. I have never seen polyps develop in the nose after this operation and washing, even in those cases which had periodically come to me for polyp removal for years previous to the washing out of their antrums. The red streaks on the

pharynx, just posterior to the posterior tonsillar pillars clear up except during acute colds, and the pharynx returns to normal. The heavy scabs and the thick skin-like coating over the pharynx and in the nasal pharynx disappear, even in patients where they have been present for years or decades. After having conscientiously washed and sprayed and swabbed post-nasal catarrh for years, never realizing that most of them come from diseased antra, there is a large degree of satisfaction in having finally got the better of them and in being able to at least discard the post-nasal syringe and the silver nitrate applicators. After operation, the antrum can easily be inspected for several days with a medium sized Killian nasal speculum inserted in the gum opening. The nose has no reactive swelling, even immediately after operation, and good nasal breathing is maintained the night following operation. The cheek is usually only slightly swollen, or there may be no swelling at all. This is particularly the case since I have adopted the plan of removing the tubes after the first twenty-four hours. The oral opening has never failed to heal within two or three weeks and one month later hardly any evidence of it remains. I still do the intranasal operation in some of my antrum cases, but of the two procedures, I regard the technique described as being the more conservative operation. There is less general reaction, the interior of the nose is better preserved, the mucous of the antrum has a better chance to return to normal.

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400 Brandeis Theatre Bldg.

CASE OF INTRADURAL CEREBELLAR ABSCESS COMPLICATED BY ACUTE LABYRINTHITIS. CASE OF LABYRINTHITIS COMPLICATING CHRONIC MASTOIDITIS.*

DR. SAMUEL J. KOPETZKY, and DR. ALFRED A. SCHWARTZ, New York.

CASE I. CASE OF INTRADURAL CEREBELLAR ABSCESS COMPLICATED BY ACUTE LABYRINTHITIS.

S. M., age 25, first came under observation in April, 1921.

Previous Otological History: Five years ago, while a soldier in France, the patient was subjected to severe exposure. At first he began to complain of frontal headaches and high fever. Then there followed pain in the right ear and spontaneous rupture of the membrane tympani. A profuse purulent discharge ensued from the middle ear which lasted twenty-five days, and then a post-auricular swelling developed. The patient was operated at a German prison hospital and after three months the ear discharge had stopped, and the mastoid wound had healed, and the patient felt perfectly well. At no time was there vertigo.

He remained well until January, 1921, when he began to complain of pain in the same ear, and paracentesis was performed. For three months following there was a purulent discharge off and on, and he complained of occasional attacks of vertigo during which external objects seemed to turn around him, and once the attack was so severe that he fell. During this time he had no fever and hearing was good. He did not veer when walking and there was no ataxia. Because of the chronic purulent otitis, he was discharged for disability from the U. S. Army.

His chief complaints, when first seen by us, were the attacks of vertigo and the foul discharge from the ear.

Otoscopic Examination: Right ear: Large marginal defect, right drum; profuse foul discharge from middle ear. Healed mastoid scar. Left ear: Normal. *Functional Tests*: Lateralization: To right. CA—right, 15; left, 30; M—right, 13; left, 13; C4—right, 12; left, 18; 64 D. V.—right, not heard; left, heard. With

*Read at the Section on Otology, New York Academy of Medicine, Nov. 11, 1921.

noise apparatus in right ear, hears in left ear. With noise apparatus in left ear, hears in right ear.

Neurological and Spontaneous Vestibular Reactions: Spontaneous nystagmus: None. Spontaneous pastpointing: None. Walking: Normal. Romberg: None. Ataxia (upper extremities): None. Pupils: Equal, react to light and accommodation. Visual fields: Apparently normal: Pelvic Girdle: Normal. Reflexes: Active and equal.

Vestibular Tests: To Test Horizontal Semicircular Canals: Turning to right: Nystagmus: 28 seconds to left, wide amplitude. Vertigo: 13 seconds to left. Pastpointing: Normal with right hand to right. With left hand off 1″ to right or left, but usually touches. Confirmed by check test. Turning to left: Nystagmus: 28 seconds to right, wide amplitude. Vertigo: 16 seconds to right. Pastpointing: Normal with left hand to left. Touches with right hand. Confirmed by check test.

To Test Vertical Semicircular Canals: Turning to right: Nystagmus: 35 seconds to left, rotary, wide amplitude. Vertigo: 21 seconds to left. Pastpointing: Normal, both hands to right. Falling: Normal, to right. Turning to left: Nystagmus: 20 seconds to right, rotary, wide amplitude. Vertigo: 18 seconds to right. Pastpointing: Normal, both hands to left. Falling: Normal, to left.

Caloric Tests, Irrigating with Cold Water: Irrigating right ear, to test right vertical canals: Nystagmus: After 60 seconds normal rotary. Vertigo: Normal. Pastpointing: None with either hand. Head back to test right horizontal canal: Nystagmus: Normal, horizontal. Pastpointing: None with either hand. Irrigating left ear, to test left ventical canals: Nystagmus: After 40 seconds, normal rotary. Vertigo: Normal. Pastpointing: None with right hand. Normal with left hand to left. Head back to test left horizontal canal: Nystagmus: Normal, horizontal. Pastpointing: None with right hand. Normal with left hand to left.

The tests show a marked impairment in hearing in the right ear, and a normal and active labyrinth. The abnormal features of the examination were: 1. On rotating to the right with head erect, he did not pastpoint properly with the left hand. 2. On rotating to the left with head erect, he did not pastpoint properly with the right hand. 3. Upon irrigating the right ear with cold water, with head erect and then backward 90 degrees, there was no pastpointing with either hand. 4. Upon irrigating the left ear with cold water, with head erect and then back 90 degrees, there was no pastpointing with the right hand.

No attempt is made here to account for the contradictory findings—why there should be normal pastpointing when the head is forward in the turning tests and off when the caloric test is done. It is noted, too, that the vertigo was normal. These findings were checked and are therefore reported.

On account of the foul middle ear discharge, and the history of vertigo and the peculiarity of the pastpointing tests—which in the presence of a normal reacting labyrinth pointed to probable cerebellar involvement—the patient was admitted to the Beth Israel Hospital service of Dr. Kopetzky on May 10, 1921.

During the week following, he occasionally complained of severe pain in the mastoid region and at times was dizzy. There were no spontaneous reactions—nystagmus or pastpointing.

On May 20, the mastoid wound was explored by Dr. S. J. Kopetzky. An incision was made at the site of the mastoid scar. In spite of extreme care, a small amount of clear fluid, apparently cerebro-spinal fluid escaped from the upper angle of the incision. When the mastoid region had been completely cleared, it was found that at the upper angle of the wound the dura was adherent to the skin for about one inch in diameter. It appeared to be the site of an old abscess cavity which had healed, and was left undisturbed. The dura covering the cerebellum projected into the wound and when this was incised, there was a flow of pus from an intradural abscess which was apparently pressing upon the cerebellum.

A small sinus at about the center of the mastoid region presented, which led to the middle ear. This tract was exposed and the antrum and middle ear was found filled with cholesteatoma. The cholesteatoma matrix was stripped as far as possible and the radical mastoid operation completed in the usual manner. On account of the operative findings, the posterior wound was not closed.

On the day following operation, the patient complained of nausea and dizziness and suffered with a severe headache. He vomited several times. The temperature arose as high as 101.8.°

On the second day after operation, the following notes were made. The pupils are regular, react to light and accommodation. There is a spontaneous nystagmus to the left, always horizontal in character. Knee jerks active. Normal plantar reflexes. No Kernig or Babinski signs. No ataxia; no disturbance apparent in equilibrium. Gait slow, no defect. Sensation—normal to touch, pain and temperature. Orientation normal, no adiodokokenesis. No disturbance in vision, except for vertigo, the room seeming to turn to

the left. He vomited several times. He lay on the left side only, and was then fairly comfortably. Vertigo and nausea would return if he lay on the right side. Lumbar puncture—albumen and globulin present; Fehlings reduced; 100 cells to c.cm.; 5 per cent polys—95 per cent monos.—no micro-organisms.

On the next day, the patient felt very comfortable and showed no evidence of cerebral or cerebellar involvement.

Convalescence was uneventful until June 9, nineteen days after operation, when a slight nystagmus was noted on looking to the extreme left. At the same time a complete facial palsy appeared—the brow on the right side could not be wrinkled, nor the eye closed. The entire right side of the face was flat and the patient could not whistle. The nystagmus disappeared and the facial palsy gradually improved and in two weeks was completely gone.

For about thirty days after the operation the patient was compelled to lie upon the left side. At first he could not turn to the right at all without extreme vertigo; this improved and in about a week he was able to turn to the right slowly without dizziness, and finally he was able to move about without discomfort.

He was discharged from the hospital on July 2, 1921. Only once, on the day following the operation, did the temperature reach above 101.° For a week after, the range was between 99° and 100,° and was normal thereafter until the date of the discharge. The mastoid wound was practically closed, and there was a slight discharge from the middle ear.

On September 4, the patient complained of rather severe vertigo and headache, and when walking felt that he veered slightly to the left. The mastoid wound was completely healed, and there was a very slight purulent discharge from the middle ear. The symptoms continued unchanged until September 8, when the following examination was made:

Otoscopic Examination: Right ear: Mastoid wound healed, slight purulent discharge from middle ear, moderate sized cavity of radical mastoid operation. Left ear: Normal.

Functional Tests: Lateralization to left: CA—right, 0; left, 30; M—right, 12 (to left); left, 12; C4—right, 0; left, 25; 64 D. V.—right not heard; left, heard; with noise apparatus in right ear; hears in left ear with noise apparatus in left ear, does not hear in right.

Neurological and Spontaneous Vestibular Reactions: Spontaneous nystagmus: Slight spontaneous nystagmus, rotary, on looking

up and to the extreme left. Spontaneous pastpointing: None. Walking: Veers slightly to left. Romberg: Sensation of swaying to right, but no swaying objectively. Ataxia (upper extremities): None. Pupils: Equal—react to light and accommodation. Visual fields: Apparently normal. Pelvic Girdle: Normal. Reflexes: Normal.

Vestibular Tests: To Test Horizontal Semicircular Canals: Turning to right: Nystagmus: 20 seconds to left—apparently slight rotary. Vertigo: 10 seconds to left. Pastpointing: With right hand, normal—none with left hand. Turning to left: Nystagmus: 16 seconds to right. Vertigo: 11 seconds to right. Pastpointing: Normal with left hand to left. Pastpoints to right with right hand.

To Test Vertical Semicircular Canals: Turning to right: Nystagmus: 20 seconds rotary, to left. Vertigo: 12 seconds to left. Falling: Sensation of falling to left, marked—no objective falling. Turning to left: Nystagmus: 15 seconds to right, slight rotary. Vertigo: 13 seconds to right. Falling: Sensation of falling to right, marked. Slight falling objectively.

Caloric Tests, Irrigating with Cold Water: Irrigating right ear, to test right vertical canals: Nystagmus: Does not increase spontaneous nystagmus—after 5 minutes. Vertigo: None. Pastpointing: None. Head back to test right horizontal canal: Nystagmus: Spontaneous nystagmus not increased. Pastpointing: None. Irrigating left ear, to test left vertical canals: Nystagmus: After 45 seconds. Vertigo: Normal. Pastpointing: Very slight to left; usually touched. Head back to test left horizontal canal: Nystagmus: Normal. Pastpointing: Very slight to left, usually touched.

Comment: That the right labyrinth is completely destroyed is obvious. The cochlea is dead and there is no response from the vestibule upon caloric stimulation. The rotation tests show a marked diminution in vertigo and nystagmus, always noted when one labyrinth is destroyed.

There was present a slight spontaneous rotary nystagmus, on looking upward and to the extreme left. On walking he veered to the left. The pastpointing was practically the same as prior to the operation. 1. No pastpointing with left hand on turning to the right. 2. Off with right hand on turning to the left. Of course, no pastpointing was to be expected on douching the right ear, as the end organ was not functionating.

From the symptoms it seemed probable that the intradural abscess was refilling.

On September 10, the entire post-auricular wound broke down and there was a profuse purulent discharge from both mastoid wound and middle ear. The symptoms immediately began to ameliorate, and very gradually disappeared. In two weeks the mastoid wound had closed, a slight discharge from the middle ear persisting.

Labyrinthine examination on November 10, showed no spontaneous nystagmus or pastpointing and the patient felt well.

Attention is directed to the expectant treatment of the acute labyrinthitis. In view of the findings in the cerebro spinal fluid, it was decided to observe the case further; and the result proved the wisdom of this course.

CASE II. CASE OF LABYRINTHITIS COMPLICATING CHRONIC MASTOIDITIS.

S. M. (Mrs.), age 25, first came under observation on September 12, 1921.

Previous Otological History: At the age of nine, there was a purulent discharge from the right ear. There is no knowledge of disease preceding the onset. Until the age of twenty, a foul odor could be detected coming from this ear, although at no time was there a visible discharge. At no time was there treatment, and the patient claims that hearing was very good.

Five years ago, the discharge suddenly became very profuse and of very foul odor. With this onset, she could not place her hands upon the object she wished to touch. There was a numbness of the right side of the face and the patient remembers that she could not whistle, but could drink without difficulty. At the same time the right hand felt numb, but could be moved easily. This attack lasted only fifteen minutes and after it the patient felt exactly as before. At no time was there nausea or vertigo, and she did not veer when walking. No ataxia persisted, and there was no facial palsy; hearing was good.

During the year following, polyps were removed from the right ear fourteen times, and the hearing became progressively worse. The foul, purulent discharge continued in spite of treatment. Since the onset five years ago, there have been attacks of vertigo occurring three or four times a week, very severe, but lasting only a few seconds. Once the attack was so severe that the patient fainted. The patient believes that the room turns to the right, but is not certain of it. Occasionally she is relieved by lying on the right side. There are severe headaches, more marked on the right side: and

she always veers to the right when walking. At the present time, the patient claims to be totally deaf in the right ear.

Three weeks ago, a discharge was noticed from the left ear. There was no pain or previous illness; no odor. She hears well.

The patient has one child; no miscarriages.

Examination: Right ear: Large marginal perforation, the entire drum, including the greater part of Shrapnels membrane destroyed. No remnant of the ossicles seen. Middle ear contains firm granulations. On examining with a blunt probe, a portion of the promotory was found dehiscent. Left ear: Small central perforation; small amount of thin purulent discharge. Functional examination: —right, 12; left, 14. C4—right, 6; left, 18. 64 D. V.—right, not There is no hearing in the right ear. The left ear shows a diminution to half of normal. Lateralization to left: CA—right, 0; left, 30. M heard; left, heard. With noise apparatus in right ear, hears in left ear; with noise apparatus in left ear, does not hear in right ear.

Neurological and Spontaneous Vestibular Reactions: Spontaneous nystagmus: Marked, to left, on looking to left. Horizontal in type. Spontaneous pastpointing: Six inches to right with right hand. Occasionally off with left hand. Walking: Veers to left. Gait unsteady. Romberg: Marked swaying to right. Ataxia (upper extremities): Slight ataxia, both hands, in finger to nose test. Pupils: React to light and accommodation. Visual fields: Apparently normal. Pelvic girdle: Normal. Reflexes: Active and equal. No adiodokokinesis. Right arm and grip weaker than left. Is more unsteady when standing on left leg than on right.

Vestibular Tests: *To Test Horizontal Semicircular Canals*: Turning to right: Nystagmus: 12 seconds to left, short, active. Vertigo: 17 seconds to left. Pastpointing: Both hands to right—the spontaneous pastpointing is increased in amplitude. Turning to left Nystagmus: 13 seconds to right—more active components than on turning to right. Vertigo: 17 seconds to right. Pastpointing: Always to right with right hand. Slightly to left with left hand.

To Test Vertical Semicircular Canals: Turning to right: Nystagmus: No nystagmus seen. Vertigo: 13 seconds to left. Falling: Had sensation of falling, not marked—did not fall objectively. Turning to left: Nystagmus: To right, 15 seconds. Vertigo: 10 seconds to right. Falling: Slight sensation of falling; did not fall objectively.

Caloric Tests, Irrigating with Cold Water: Irrigating right ear, to test right vertical canals: Nystagmus: None. Vertigo: None.

Pastpointing: Off, both hands. Head back to test right horizontal canals: Nystagmus; None. Pastpointing: Off, both hands. Irrigating left ear, to test left vertical canals: Nystagmus: After 35 seconds, marked, rotary to right. Vertigo: Normal. Pastpointing: Normal, to left, both hands. Head back to test left horizontal canal: Nystagmus: Normal, horizontal to right. Pastpointing; Normal, to left, both hands.

Comment: The destruction of the labyrinth is shown by the following:

1. The function of the cochlea is completely destroyed.

2. The labyrinthine tests show a complete absence of response from the right side, for (a) The turning tests show a marked diminution in vertigo, with less than half the normal nystagmus. (b) The caloric tests elicit no response from the right ear.

There are, however, several symptoms which are not explained by the destruction of the labyrinth.

1. There is a marked spontaneous nystagmus to the left, especially marked on looking to the left.

2. There is spontaneous pastpointing with the right hand to the right, and the patient pastpoints off with the left hand.

3. On walking with the eyes closed, she veers to the left, although she claims that she always walks to the right.

4. Marked Romberg—sways to the right.

5. The right arm seems weaker than the left.

6. The right grip seems weaker than the left.

7. She is more unsteady when standing on the left foot than the right.

These symptoms, though in places apparently contradictory, together with severe headache, led to the tentative diagnosis of cerebellar abscess, and operation was advised, but refused. The case is reported, however, because of the evidence of the destruction of the labyrinth. The patient disappeared from observation and no smear or culture was obtained; and although the case is suspicious of tubercular otitis, because of the history and physical findings, there is no proof of such being the case.

AERATION OF THE POSTERIOR ACCESSORY SINUSES IN ACUTE OPTIC NEURITIS.*

DR. LEON E. WHITE, Boston, Mass.

The pleasure in presenting a paper before this distinguished Society is mixed with fear and trepidation lest these meager gleanings, from a study of infections in the accessory sinuses as a cause of optic neuritis, may not be sufficiently worth while.

Beginning this work some ten years ago, I was stimulated to continue by the remarkable results obtained in early aeration of the sinuses on the one hand, and on the other by the pitiable and pathetic spectacles of permanent blindness which might have been prevented. During these years there have been times when adverse criticism and scepticism have tempted me to abandon the field, but the continued expressions of disbelief as to the advisability of operating unless marked pathology was evident in the nose, convinced me that it was my duty to stick to this investigation. To the kindness and encouragement of Dr. Mosher, I am deeply indebted. As chief of the Nose and Throat Department of the Massachusetts Charitable Eye and Ear Infirmary, he has permitted me to handle the cases referred from the ophthalmic side for this condition which has enabled me to study a large number and to follow them year after year. By thus keeping in touch with these cases, it is possible to check up the early results and to determine whether the lesion is peripheral or central. Dr. Quackenboss and Dr. Spalding, the ophthalmic chiefs, and Dr. Verhoeff, the pathologist, have rendered great service, as well as many others on both the aural and ophthalmic staffs.

Certain phases of this subject I have already considered, such as the anatomical relations of the optic nerve; the literature; the diagnosis; the prognosis; the etiology; pathology, etc. In each paper there has been a report of the cases followed since the preceding one, so that the one appended to this article is my thirty-fourth.

A brief resumé of the work done to date may be of interest. The relations of the optic nerve to the accessory sinuses have been carefully worked out. In 1886 Berger and Tyrman reported their

*Read at New York City on October 26, 1921, before the joint meeting of the Sections of Ophthalmology, and of Rhinology and Laryngology, New York Academy of Medicine.

findings in the examination of the differences in the partition wall between the optic nerve and sphenoidal sinus and noted that the bony wall separating the nerve from the sinus was frequently of only tissue paper thickness. Onodi, in 1908, said:

"For ten years I have been investigating the most delicate construction of the accessory cavities and the relation of the optic nerve to them." His work was so thoroughly done that later research has been largely a confirmation of his findings. In 1911 he published an atlas with natural size plates showing thirty-eight different morphologic findings on the relations of the optic nerve. Let me quote one paragraph:

"Our observations have shown that the wall between the last ethmoid cell and the canalis opticus is nearly always as thin as tissue paper; dehiscences in the walls of the accessory cavities have been found, there the diseased mucosa may come into direct contact either with the dura mater or the optic nerve sheath."

Normally, according to Loeb, "the optic nerve may be described as passing externally from the chiasm along the roof or lateral wall of the sphenoid and in close relation with the ethmoid labyrinth only at the posterior external angle of the last cell. * * * Where this normal relation exists there is only the slightest possibility of any danger to the nerve in suppuration confined to the ethmoid cells. * * * But where the posterior ethmoid cell replaces a portion of the sphenoidal sinus, the optic nerve runs close to, and along the external wall of this ethmoid cell (as in two of the thirty specimens he studied) and its vulnerability is correspondingly heightened in view of the greatly increased portion exposed."

At the 1921 meeting of the American Medical Association, Dr. Schaeffer gave a lantern demonstration of these relationships and showed many beautiful and instructive specimens. From his book on "The Nose and Olfactory Organ" the following is quoted:

"It is essential that the intimate anatomic relationships which exist between the paranasal sinuses and the optic nerve and commissure be understood by ophthalmologists and rhinologists. It is established that disease of the paranasal sinuses may lead to an optic neuritis, even to blindness. Of the paranasal sinuses, the sphenoidal and the posterior ethmoidal especially concern us in this connection. * * * Indeed, for a considerable distance from the eyeball, the optic nerve is so far removed from the paranasal sinuses that very intimate relationship is precluded by the intervention of a considerable mass of orbital fat. However, as the optic nerve

approaches the orbital apex and passes through the optic foramen to the optic commissure, very intimate relationships exist between some of the paranasal sinuses and the nerve and its commissure. * * * The optic nerve pursues a course ventralward from the optic commissure along either the roof or lateral wall of the sphenoidal sinus. Frequently a posterior ethmoidal cell is more or less intimately related as well. After the optic nerve passes beyond the vicinity of the posterior ethmoidal cells, it diverges more and more from the ethmoidal field and is no longer in intimate relationship with the other ethmoidal cells."

From the investigations of these men you will see it has been demonstrated that the optic nerve may be in close relationship to the sphenoidal sinus and the posterior ethmoidal cell. To these and to no other of the accessory sinuses. The sphenoids and posterior ethmoids are therefore the only sinuses in intimate relation with the optic nerve, and to reach the tissue adjacent to the nerve the direct and logical route would be through these structures and not through the entire ethmoidal labyrinth.

The literature on accessory sinus blindness is voluminous and dates back to the observations of Beer in 1817. He says in an article on "Vicarious Blindness from Suppressed Snuffles, Without Evident Accumulation of Mucous in the Frontal Sinus," "that the recognition of this form of amaurosis is greatly facilitated by a history of a severe and suddenly suppressed cold in the head immediately preceding the ocular complication." He further says that "the cases all do well if one is able to re-establish a copious discharge of mucous from the nose."

In 1886 Berger and Tyrman gave a brief review of the previously reported cases of blindness, some twenty-six in number, arising from sphenoidal disease.

In 1915, Stark collected eighty-eight cases from the literature. Since then the number reported has rapidly increased, but time only permits mention of some of the contributors: Onodi, Sluder, Loeb, Knapp, Holmes, de Schweinitz, de Kleyn, Berger, Van der Hoeve, Halstead, Stark, Beck, Skillern, Vail, Posey, Bordley, Smith, etc.

While some writers recognize that it is not always possible to diagnose the nasal condition responsible for the optic neuritis, and have advised operating even if the nasal findings were negative, the general impression prevails, I believe I am justified in saying, that

a purulent infection, even if unrecognized, must exist. So persistent is this belief that I am not infrequently told the accessory sinuses were eliminated because the roentgenograms were negative.

The diagnosis: This is naturally of major import. The symptoms in the ordinary case of acute optic or retrobulbar neuritis are quite typical. Associated with the losses of vision, there may be discomfort about the eye or lameness on moving it. Occasionally there is slight exophthalmos, this condition indicating an inflammatory process in the orbit. Pupillary changes and ptosis are found occasionally. There is frequently a central scotoma for colors and contraction of the fields. The value of quantitative perimetry has been emphasized by Walker and is of assistance in making a differential diagnosis but as many patients have not even light perception, these charts are not always obtainable. The patients frequently remember that things at first were blurry when looking straight ahead but comparatively clear when looking sideways. Changes in the fundus are of great value, varying as they do from normal to a commencing pallor in some cases and in others to a marked redness and swelling about the nerve head engorgement and tortuosity of the retinal veins. It is essential that these inflammatory swellings be differentiated from those due to intracranial pressure.

The anterior portion of the nose may, and frequently does, appear practically normal. I believe the *one vital point* to determine in the nasal examination is the *size* and *position* of the middle and superior turbinates. Do they block the ventilation of the posterior sinuses? Is there impaired aeration? It takes but slight obstruction to interfere with the ventilation and the mistake is frequently made of expecting to discover marked changes.

There has been much controversy over the term so frequently used, "negative nasal findings." While pleading guilty to the use of this term, it was done to impress upon the rhinologists that but little was evident on inspecting many of these noses; also because several of my cases had been examined and reported as negative even when to me they presented evidence of blocking. One case in particular had a marked deflection of the septum which wedged the middle turbinate so firmly between the ethmoidal labyrinth and sphenoid that there could be no question whatsoever of its blocking, and yet because pus was not evident and the X-rays negative, the report was made that there was no accessory sinus involvement. This man developed optic atrophy and as the vision continued to fail he was referred to me a year later, when I removed

the middle turbinate. Following this, there was improvement, but only slight (from 20/200 to 20/100).

The term "negative nasal findings" does not mean that the nose *is* normal. Normal noses do *not* produce optic neuritis, but optic neuritis can frequently be produced from noses which to some examiners *seem* normal. It is doubtful if any other field in surgery requires greater discrimination in making a differential diagnosis. I should hesitate to operate on any case where ventilation was possible by shrinking the tissues in the region of the posterior sinuses.

One can generally determine by the use of cocain and adrenalin whether there is a temporary swelling or a chronic enlargement of the middle turbinate. Deflected septi with crowding of the chronically enlarged middle turbinates are the most usual findings. The following is quoted from Stark, who expresses well the thought I wish to convey:

"From a nasal standpoint we must not expect to find the common symptoms of sinus infection, pus, polypus, history of nasal discharge, etc., as we are dealing with a closed sinus; otherwise we should not have pressure. * * * The deflected septum and middle turbinate tightly pressed against the lateral wall should always be suspected."

Over half the cases give a history of a recent coryza or a prior influenza, but in a few there is no history of any nasal infection. Roentgenograms are usually disappointing. Occasionally slight blurring but practically never marked evidence of sinus disease.

Dr. Macmillan, the radiologist at the Infirmary, summarized the findings as follows:

"The radiographic examination in this group of cases was rather disappointing from the standpoint of one seeking definite pathology. In a number of instances, no clouding could be made out in the sphenoidal or ethmoidal cells; in others a slight blurring of the cell partition was evident, while in none of the cases was there definite demonstrable pathology. There was no evidence of involvement of either the frontal sinuses or the antra in any instance."

The diagnosis many times must be made largely by exclusion. In all cases, the patient should undergo a thorough physical and neurological examination. One should consider in turn, blood, urine, teeth, tonsils, hysteria, pellagra, lues, tobacco, alcohol, lead, arsenic, quinin, etc. While investigating, *time* is a very important element and it is essential that needless delay be not encountered. Practically all necessary tests can be made within forty-eight hours.

Pituitary disease, brain tumor and multiple sclerosis, while not usually producing such *sudden* loss of vision, must always be borne in mind. As a matter of routine, the various cranial nerves and lobes of the brain are tested. Not a few cases of brain tumor, Dr. Cushing informs me, have had various nasal operations before they were correctly diagnosed. This careful investigation has prevented unnecessary nasal operating in several cases I have seen in consultation. One should systematically go over each case before operating and convince himself that the eye condition in all probability originates from the accessory sinuses.

Etiology: The earlier writers considered the mere presence of pus in the sinuses the all sufficient explanation for the disturbance about the optic nerve, and while this may undoubtedly be the cause in some instances, it is by no means the usual or only one. The nerve is ordinarily protected by the barrier thrown out by inflammatory processes so that it is rarely involved unless through some anatomical abnormality. In only a small percentage of my cases has pus been found. Hyperplasia has been emphasized by others as the chief cause, and while hyperplastic tissue may involve the posterior sinuses, it has seemed to me from my study of the sections and cases that it would better be considered a predisposing factor rather than the principal etiologic condition. Hyperplasia undoubtedly renders the sinuses more vulnerable. The etiology seemed to be explained in many of my cases by the size and position of the middle and superior turbinates; poor ventilation and faulty drainage appeared the more important predisposing factors. The theory advanced by me in a paper read before the Triological Society in 1921 is that these superior turbinate structures high up in the roof of the nose gradually become enlarged and obstructive. There is an increase in the basal connective tissue, *i. e.*, a certain amount of hyperplasia, so that eventually the posterior sinuses are practically blocked. Then some infection takes place in these sinuses which, if not already occluded by the hypertrophied turbinates, most readily become so by a very slight increase in their size. Then an inflammatory process commences. This is generally of the exudative but non-suppurative type. The swelling and infiltration incident to it spreads by continuity of tissue to the optic nerve, or (if the sinuses are completely shut off) there may be a closed empyema with infection to the nerve through the circulation—not in all cases, of course, but in those which, through some anatomical peculiarity, are rendered especially vulnerable. The infection may be confined to the region superior and

posterior to the middle turbinate, thus explaining why so little is seen on inspection and why roentgenograms show but slight, if any, changes, *i. e.*, only what would be expected from an acute non-suppurative inflammatory process.

Pathology: While it would be most gratifying to have some comprehensible explanation of the pathology in the posterior sinuses, it is vastly more imperative that the results of appropriate treatment be given just weight. The unfortunate victim with optic neuritis wants his vision. If aeration of the posterior sinuses offers the best chance for recovery, why not aerate, even if one fails to find pus or marked disease? Later, if need be, let us differ as to why recovery takes place so speedily when so little is discoverable at operation. As Loeb says, "the ready recovery * * * is as convincing of their nasal origin as anything could be, short of autopsy findings." Those demanding an explanation of the pathology by the examination of the tissue, either macroscopically or microscopically, are frequently bound to be disappointed. Dr. Jonathan Wright's concise summary is most apropos: "Endless talk and circumstantial evidence of the symptoms cannot decisively settle anything and there is little hope of getting the convincing pathological material in which infection or spreading inflammation can be traced objectively."

While not a *pathologist,* I have undertaken the consideration of the subject under five heads for convenience, *viz.*: 1. Direct extension; 2. Toxemia from some infective process; 3. Bacteremia or focal infection; 4. Hyperplasia; 5. Anaphylaxis.

1. *By direct extension,* infections in the posterior sinuses extend by continuity of tissue to the optic nerve. These infections are frequently non-suppurative, hence the negative roentgenograms. The question is often raised, "Why is there loss of vision with so little evidence of pathology in the nose, and yet rarely any visual disturbance where there is marked disease?" The answer is that in these chronic cases nature probably walls off the eye, the optic nerve, and, in fact, the body from the source of infection. When an acute infection occurs in the sphenoid or posterior ethmoid, the sinuses probably become closed and there follows an almost immediate invasion of the tissue about the optic nerve. When, however, there is an infection in a more remote sinus, it may become walled off before the nerve is reached. Should the infection persist, there may be forced into the system a certain amount of bacteria and toxins, which in turn can produce an optic neuritis. While chronic infections may occasionally involve the optic nerve, the

acute and subacute infections with but little discoverable evidence in the nose are of far greater consequence. The fact that it is so easy to overlook these conditions leads me to dwell upon their importance:

2. *Toxemia*: It is conceded that retrobulbar and optic neuritis can be caused by alcohol, lead, tobacco, quinin, optochin, arsenic, lues, etc., so that reasoning by analogy there is little doubt but that toxins originating in the accessory sinuses, or for that matter anywhere in the body, may have similar action on the optic nerve. The onset is usually less violent than where there is a direct extension and is more apt to be a causative factor in chronic types. It is quite generally conceded that any pus focus within the body may be a factor in these vision cases, and while this paper deals only with the accessory sinuses, other possible sources must not be overlooked.

3. *Bacteremia*: It has been demonstrated by Billings that infectious micro-organisms may be carried in the blood stream or by the lymph channels from the foci of infection in the teeth, tonsils and accessory sinuses to the terminal blood vessels in various regions of the body. He has shown how the inoculated blood vessels become more or less occluded by endothelial proliferation and leukocytic infiltration and that the bacteria escape through the vessel walls into adjacent tissue, so there would seem little doubt but that bacteria within the accessory sinuses may also travel via the blood stream and lymph channels to the optic nerve.

It was probably due to bacteremia that the vision in one of my cases (thirty-three) remained at a standstill so long. This patient when operated on three weeks after the onset of the retrobulbar neuritis was only able to count fingers at six feet and the nerve head was turning white. Within forty-eight hours the vision had doubled and the discomfort and lameness about the eye which had been marked, disappeared. Following a secondary hemorrhage during which the posterior nares were plugged, there was a very severe infection in both middle ears and mastoids. This continued four weeks. One side recovered under treatment but it was necessary for me to open the mastoid on the other side. During these four weeks the vision remained stationary but following the draining of the mastoid it commenced to improve again and when last seen, some three months later, was 20/60.

4. *Hyperplasia*: As a predisposing factor, hyperplasia is probably of considerable importance. It undoubtedly renders the ac-

cessory sinuses more vulnerable. Hyperplasia plus infection and direct extension to the optic nerve is probably of far greater consequence than the mere fact that the tissue has become hyperplastic.

5. *Anaphylaxis*: In a paper read at the 1921 session of the American Medical Association, Stark advanced the theory that there is a "sensitization of the tissues of both the sinus and the orbit by the bacterial proteins, producing an allergy, resulting in a localized anaphylactic reaction each time the individual comes in contact with a fresh infection of the same bacteria in the nose, and possibly in other parts of the body. For that reason, many of these cases give a history of attacks resembling hay fever, or acute coryza, shortly previous to the eye trouble." While I have had a few cases presenting symptoms similar to those mentioned by Stark, it had not occurred to me that this would satisfactorily explain the pathology. It would, of course, explain the negative roentgenograms and the meager findings on opening the sinuses. It is, I believe, a valuable suggestion. There certainly seems to be a similarity between some of these eye conditions and certain anaphylactic reactions comparable to asthma and hay fever. As sinus infections frequently cause asthma, it is conceivable that they might also produce engorgement about the optic nerve. Further investigation along this line I sincerely hope may clear up many points which are but imperfectly understood at present.

Just a word as to what we are doing in pathology and the findings. The middle turbinate and the tissue from the sinus walls are placed in separate specimen bottles and after being carefully marked are sent to the laboratory for study, together with cultures and smears from the sphenoid. Sections from ten cases I have brought with me which may be examined by those interested. I suspect, however, that most of you will prefer to read Dr. Jonathan Wright's interpretation of these slides, which is included in my paper on Etiology and Pathology, shortly to appear in THE LARYNGOSCOPE.

Dr. Martland of the Newark City Hospital also looked over the slides and summarized his findings as follows:

"Most sections show intact mucosa; in many places it seems to be edematous and hyperplastic. Submucosa varies all the way from normal to areas containing numerous small mononuclears, practically no polymorphonuclears; some sections show a considerable number of eosinophiles, indicating low grade chronic infection. Submucosa is often edematous; in some places there is questionable

rarefying osteitis. Many sections show considerable dilitation of submucosal vessels (acute hyperemia) and undoubtedly represents low grade chronic inflammation, which is non-suppurative.

Conclusion: It is quite possible in non-suppurative inflammation that edema of mucosa and submucosa with acute hyperemia of vessels, etc., may produce more pressure than a suppurative process, in which the pressure is often relieved by the breaking down of the tissues."

The prognosis depends largely upon the duration and extent of the loss of vision, the condition of the fundus and the virulence of the infection.

1. As to the *duration,* I have endeavored to determine how long an interval could elapse before there would be danger of permanent impairment of vision. In the thirty-four cases tabulated, four (all of short duration) recovered under local treatment. Seven operations were performed in the first week and practically normal vision obtained in all. Of the six patients operated upon within two weeks, two recovered with normal vision, three with vision 20/20, but with slight pallor of the nerve, and one with vision of fingers at three feet with optic atrophy. Of the six cases in which operations were performed between the second and fourth weeks, normal vision but with some pallor was obtained in one; improvement in all the others. Four patients were operated upon in the second month. One obtained normal vision, another 20/20, but with some pallor. In the other two, optic atrophy with no improvement in one, and but slight improvement in the other. Of the six cases of over two months' duration, there was no improvement in three, and in the others it was so slight as to be almost negligible. In one case of four years' duration there was complete optic atrophy and of course no operation.

From the foregoing summary it may be said with some degree of assurance that unless a case shows improvement under treatment within a week, there is danger of permanent impairment of vision, unless pressure on the nerve can be relieved. In cases of more than two months' standing little can be expected, except possibly that the progress of the disease may be checked if due to some sinus infection.

2. As to the *degree of loss of vision*: In the seven cases in which there was complete blindness, two (of eight and ten days' duration) returned to normal but pallor of the nerve remained. In one of four weeks' standing, fair sight was established. Un-

improvement in three, while in another fingers could only be counted at three feet. Thus the demand for early operative interference in total loss of vision is more imperative than when the loss is but partial.

3. *Condition of the fundus.* When the nerve appears normal one might, with safety, delay operating much longer than where there is increasing engorgement, or commencing pallor. It has seemed to me so imperative that the fundus changes should be watched from day to day that I have spent many hours during the past year trying to train my eye to this task.

4. The *virulence of the infection.* As in all the other types of infection, so in that producing optic neuritis, the micro-organisms differ greatly in virulence. When the infection is of the virulent type, there is probably considerable exudate about the nerve or even within its sheath. The optic nerve, as you know, is really not a nerve but a part of the brain. It is easily destroyed and does not regenerate. Parsons says of it:

"The so-called optic nerve together with certain parts of the retina, constitutes a lobe of the brain, and has therefore the characteristics of the central nervous system. Hence the nerve-fibres are devoid of a sheath of Schwann and the interstitial substance is neuroglia."

Whenever there is an exudate we have to contend with subsequent shrinking, which may destroy the function of the nerve even though the pressure is relieved. This helps to explain why there are not complete recoveries in some early operations. Several of my cases showed pallor of the disc, which probably resulted from this exudate. One of only ten days' duration had marked optic atrophy. The virulence of the infection in another case (thirty-one) was so marked that had the accessory sinuses not been promptly drained the vision would probably have been permanently lost. The case is also interesting from the fact that she was seen from the very onset of the trouble, being referred by Dr. Quackenboss on March 15, 1921, with diagnosis of optic neuritis—right. History: fair general health, but rather tired; has been subject to colds, has had one for four or five days, accompanied by pain about the eye for the past twenty-four hours, so that when seen she looked extremely ill. Eye was sensitive to light and on movements and pressure. Vision when first seen was 20/20. The right middle turbinate was somewhat enlarged and the septum deflected to that side. No secretion was seen within the nose but there was a marked pharyngitis. Transillumination was negative.

Roentgenograms showed right posterior ethmoids slightly clouded and infection about one tooth which was later extracted. The physical, neurological and Wassermann examinations were all negative. Two days later the patient's vision was 20/40, central scotoma for colors. The following day there was an increase in the neuritis; vision 10/100. In view of the negative neurological examination, the rapidly diminishing sight and the increase in the inflammation of the optic nerve, it was deemed advisable to open the accessory sinuses at once. Under general anesthesia the right middle turbinate was removed, the sphenoid opened and the posterior ethmoid uncapped. The tissue was somewhat inflammed but no pus was seen. The lining wall of the sphenoid was little, if at all, changed. Cultures and smears were made and specimens saved for study. On the day following the operation the patient felt considerably relieved and the eye was less blurry. This lasted but a few hours, then there was a rapid recurrence of the blurring, probably due to an acute coryza or some post-operative infection, and the vision continued to fail, so that a week after operation fingers could only be made out at three inches and the edges of the disc were practically obliterated, some exudate, small blood vessels engorged. Two weeks after operation, the swelling in the nose had subsided and the discomfort and blurriness were alleviated; counted fingers at eight feet; four days later fingers at twenty-five feet. Within a month the blurriness had practically disappeared and the outline of the disc was sharply defined. Vision was 20/60. Slight pallor of the nerve was noted two weeks later, vision 20/30. In six months it was 20/20. The smear from the sphenoid showed only blood and a few epithelial and pus cells. In one of the culture tubes there were three colonies of diphtheroid bacilli. Five specimens from the middle turbinate and sinuses were examined by Dr. Jonathan Wright, who reported as follows:

"(a) Right middle turbinate. Soft parts not especially altered. Some hyperplasia of fibrous tissue near the bone and a very moderate degree of increase in the cellular activity along some bone edges—blood vessel walls somewhat thickened. *Chronic inflammation of deeper elements of the mucosa and of the bone.*

(b) Another fragment of right middle turbinate. Thinner section—more impressed with bone involvement. Fibrous hyperplasia quite evident.

(c) Sphenoid. Specimen very small. Rarefying osteitus along some edges rather marked, but soft parts are largely lacking—not very satisfactory.

(d) Ethmoid. More tissue, but no epithelium. Connective tissue near bone rather damaged by decalcifier, but intense nuclear infiltration and bone change marked as in (a)—more of it, 0 im. 1/12 not very satisfactory. Section thick and cellular changes not very distinct, but one gets the impression of much cellular infiltration. Rather an acute process involving bone.

(e) Post ethmoid. Narrow long strip of bone. Nothing to add to (a)."

THE TREATMENT OF OPTIC NEURITIS.

What is the appropriate treatment for a case with sudden loss of vision probably due to accessory sinus infection? It is known that some recover spontaneously, while others untreated go on to atrophy. To operate in every instance would cause criticism, especially from those who claim that a large proportion recover spontaneously, but on the other hand permanent blindness may follow by delaying operation. Pus has only occasionally been found in the sinuses I have opened, and yet improvement usually commenced within forty-eight hours. It seems to be aeration or ventilation that is required, rather than drainage or removal of diseased tissue. Hence, the general statement may be made that if aeration can be established, the patient has been given appropriate treatment.

In speaking of the etiology, mention was made of the theory that either acute swelling or chronic enlargement of the middle and superior turbinates seemed to be the explanation in many cases. If this is a fact, relief should be obtained either by remedies causing acutely swollen turbinates to subside, or by their removal when chronically enlarged. Under local treatment, several patients have recovered, and it is in the class of acutely swollen middle turbinates that most of the spontaneous recoveries belong. As previously mentioned, one can usually determine how much the tissue will contract, and as to whether or not the superior meatus can be sufficiently opened to furnish the requisite ventilation. If, after cocanization, the turbinate is found to hang free, the case will probably recover under local treatment. While, on the other hand, should the middle turbinate after cocainization still obstruct the aeration (*i. e.*, be wedged between the ethmoid wall and what frequently is found, a posterior deviation of the septum to the affected side) then the chances are not so good for recovery under local treatment, and it may be necessary to establish aeration by at least the removal of the middle turbinate and possibly the opening of the sphenoid sinus and the pos-

terior ethmoid cell. As far as I have been able to determine, any process by which a chronic thickening of the turbinates (hyperplasia if you wish) is brought about, has probably produced some change in the linings of the accessory sinuses. I have, therefore, usually made it my practice after taking out the middle turbinate to remove the front wall of the sphenoid and uncap the posterior ethmoid cell. The other ethmoids, unless diseased, are not disturbed. If teeth or tonsils show infection, they are removed, and if an antrum is suspected, it is washed out and if pus is found, it is thoroughly opened. The more I study along this line the more this conservative method appeals to me. While on several occasions I have done a more extensive ethmoid operation, I do not now think it was necessary. Aeration of the sinuses adjacent to the optic nerve seems to be all that is required, the function of the nose is little, if at all impaired, and the procedure is less hazardous than when a complete ethmoid exenteration is performed. While to the skilled operator these radical procedures are of minor concern, the patients do not always fall into his hands. As fatalities have been reported following these extensive operations on the ethmoid labyrinth, it has seemed best to advocate a method having the advantage of being both simple and safe.

It may be superfluous to mention the operative procedure, but if I do not, some one is sure to ask; so the following is taken from a recent article of mine in THE LARYNGOSCOPE: Remove as much of the middle turbinate as is necessary to gain access to the sphenoid, by incising it below and anteriorly with a Sluder knife, then severing its outer attachment with middle turbinate scissors, followed by snare, and finally removing all fragments, and occasionally portions of the superior turbinate with biting forceps. It is especially important to freely expose the front wall of the sphenoid. Then the Sluder's sphenoid knife, with point downward, is passed along the cribriform plate until the front wall of the sphenoid is reached high up. Downward pressure easily forces the knife through this anterior wall. By this method one is working away from the brain and never toward it. With two or three strokes downward and then two or three outward, the sphenoid is sufficiently opened to permit inserting an antero-posterior pair of biting forceps with which the anterior wall is quickly removed. The posterior ethmoid is uncapped with a curet. Don't meddle with the lining membrane of either the posterior ethmoid or sphenoid.

As to post-operative complications. There have been no fatalities. Three secondary hemorrhages have occurred, two were controlled

by packing in the nose, as the third might probably have been. This is the mastoid case already mentioned in speaking of bacteremia. The patients are usually not upset by the operation and remain in the hospital but two or three days. Local anesthesia can be used if one so desires, but ether, with patient in sitting position, is usually preferable. The results have already been summarized under prognosis and it but remains to add in conclusion that my early operations have been uniformly successful. Cases of over two months' duration, however, have been benefited but little. One cannot draw sharp lines on the basis of time only. Each case must be considered on its own merits. Some with progressive loss of vision, even when there was marked pallor of the nerve, have been slightly benefited. Complete optic atrophy is, of course, hopeless.

Case 34: E. A. H., a schoolboy of 15, was referred on May 18, 1921, by Dr. William N. Souter, with diagnosis of optic neuritis, right. Patient in fair health, but rather backward; is subject to frequent colds and occasional sore throats; right eye has never focused; had severe cold a month ago. Eight days ago pain commenced in the right eye, shortly followed by almost complete loss of vision. Finger movements on temporal side close to eye when first examined. Eye sensitive to pressure and on movement. The boy had large unhealthy tonsils and adenoids. Septum was fairly straight. The right middle turbinate was of large size and shrunk but little on cocainization. It seemed without question to obstruct the posterior sinuses. Some muco-purulent secretion beneath both middle turbinates. Dr. Macmillan reported on the roentgenograms as follows: "All sinuses appear clouded, suggestive of an acute pansinusitis. Ethmoids in particular appear to be involved, the left perhaps more than right. Stereoscopic lateral examination shows sphenoid is large, but does not appear to be clouded." Dr. Vail examined him and reported: "Convergent squint ou. External recti can function. Left pupil larger than right. Pupil react to consensual reflex. Sluggish with suggestion of hippus on flashing light into right eye and exam. reaction of left. *Fundi*: o.d. disc pale, outline blurred, vessels not remarkable. o.s. disc pale yellow, outline sharp. Pigment not marked. *Visual field* on rough examination in right eye confined to upper temporal quadrant. *Vision*: Sees light shadows with right eye; left eye apparently normal. *Cranial nerves:* Normal. Patient right handed. No aphasia or cerebral disturbance. No spontaneous nystagmus nor Romberg. Knee jerk lively and equal. *Impression:* No evidence of any intracranial lesion." The other examinations being negative and the find-

ings in the nose so positive, there was no hesitation in advising immediate operation. On May 20, the tonsils and adenoids were removed, the right middle turbinate taken out, and the posterior ethmoid cell uncapped. As the opening into the sphenoid seemed of good size, it was not enlarged. There was an almost immediate improvement in vision, so that within three days he could count fingers at one foot. Two months later, Dr. Souter reported that the patient "shows excellent progress. Vision with correction 6/12 plus, fields practically normal, though disc shows some pallor." Dr. Verhoeff reported as follows on the middle turbinate specimen:

"The mucosa shows a marked infiltration with chronic inflammatory cells, plasma cells greatly predominating. There are, however, small foci composed exclusively of lymphocytes. Pus cells are practically absent. There is no tendency toward polypoid formation. The deeper tissue is normal."

SUMMARY.

The optic nerve is only in close relationship to the sphenoidal sinus and the posterior ethmoidal cell. In order to reach the tissue adjacent to it, the direct and logical route would be through these structures and not through the entire ethmoidal labyrinth.

The literature dates back a little over one hundred years and the general impression obtained from it is that purulent infections, even though unrecognized, must exist.

The diagnosis can sometimes be made almost from the symptoms, while at other times it can only be determined after the most careful and painstaking study. Roentgenograms are usually disappointing. Great care must be taken to exclude brain tumors.

No one etiological condition is responsible for all cases. While puruient infections may account for a few, there are many in which the infection is non-suppurative. Poor ventilation and faulty drainage are all important predisposing factors. The size and position of the middle and superior turbinates are of great importance in aeration of the posterior sinuses.

Pathology: 1. Direct extension; 2. Toxemia; 3. Bacteremia; 4. Hyperplasia; 5. Anaphylaxis.

1. By direct extension acute and subacute infections spread by continuity of structure to the optic nerve.

2. Toxemia. Toxins originating in the accessory sinuses may involve the optic nerve.

3. Bacteremia. Micro-organisms may be carried in the blood stream or lymph channels from the sinuses to the optic nerve.

4. Hyperplasia as a predisposing factor is of considerable importance, as it tends to render the accessory sinuses more vulnerable.

5. Anaphylaxis. There seems to be a similarity between optic neuritis and certain anaphylactic reactions comparable to asthma and hay-fever. As sinus infections cause asthma, it is conceivable that they might also produce engorgement about the optic nerve.

The prognosis depends on the duration and extent of the loss of vision, the condition of the fundus and the virulence of the infection.

Treatment. The important thing is to establish aeration and not to remove diseased tissue. Some will recover under local treatment. In others a semi-radical sphenoid operation is advocated.

Complications. No fatalities. Three post-operative hemorrhages. In one, middle ear infection followed the post-nasal packing.

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THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

January 13, 1922.

Continued from page 326.

DR. KERRISON expressed his interest in the paper and in the remarks of Dr. Dench and Dr. Dean. During the last two years there had been a great deal of discussion as to whether in the presence of a dead labyrinth preceded by labyrinthine symptoms the radical operation should be performed without the draining of the labyrinth, and the consensus of opinion seemed to be veering to a greater conservatism in regard to opening the labyrinth; but it has not yet been possible to differentiate the cases or even to determine the different types. The conditions so fairly stated by Dr. Dean would explain some of them, and the remarks which he had made, if elaborated in the form of a paper, would add greatly to the understanding of the subject and would provide a working hypothesis for discussion.

In regard to draining or opening a dead labyrinth at the time of a radical mastoid operation, perhaps each case should be decided on its merits. Whenever, however, there is any reason to suspect an active labyrinthine lesion or even an old labyrinthine suppuration in the slightest degree likely to be rekindled by the mastoid operation, surely the labyrinthine operation is the safest thing to do.

As regards operating upon the labyrinth during the acute stage of a labyrinthitis, we are at sea. Whatever we may do in a particular case, *i. e.*, whether we do or do not operate, may prove wrong as judged by the results. I personally feel that in any considerable series of cases, more lives will be saved by abstaining from all surgical intervention until all labyrinth symptoms have disappeared, than by operating during the acute stage.

As regards lumbar puncture during an acute labyrinthitis or even during the chronic stage, certain facts should be kept in mind: Babmski's experiments have quite clearly shown that lumbar puncture is very frequently followed by symptoms clearly induced by diminished intralabyrinthine pressure; this reduction of intralabyrinthine pressure can only be brought about by the withdrawal of labyrinthine fluid, however small the amount, into the subarachmoid space, and it should be clear that this may initiate a meningeal infection. It would seem to me, therefore, that lumbar puncture during a suppurative labyrinthitis should not be done as a routine measure, but only when meningeal symptoms are present and call for the definite data which this procedure provides.

DR. WHITING said that he had read Dr. Smith's paper and cordially endorsed the attitude taken in regard to the various problems connected with labyrinthine conditions. He did not think that any labyrinth which is functioning should be entered under any conditions. The conditions requiring operation: A dead labyrinth without manifest disturbance does not call for operation, except where having undertaken to do a radical mastoid there is found obviously some continuation into the labyrinth, etc. Under such circumstances, as Dr. Smith had suggested, only such operation should be done as was of the most conservative nature.

Regarding the third class of cases, one should refrain from operating except in cases as indicated by the findings of the spinal fluid where there is an active process instituted; if there is not such a process, more harm is done by operation than by refraining. As he understood it, this was the attitude taken by Dr. Smith, and he was glad to endorse that position.

DR. J. MORRISSETT SMITH, concluding the discussion, said that the hydrops and serofibrinous types of which Dr. Dean had spoken would come under the class of localized labyrinthitis. The fact that the labyrinth was still active showed that there was not a diffuse condition there, although it might at any time become diffuse. Where the condition becomes purulent, we immediately get a dead labyrinth.

As to whether one should do a radical mastoid alone where there is a dead labyrinth and no symptoms, that brought up a very interesting question. It is very important, as soon as it has been discovered that there has been trouble in the labyrinth, to have a spinal puncture. If the spinal puncture is normal and the patient has no symptoms, it is fairly certain that whatever infective process may have been present, has subsequently been walled off from the meninges. He agreed with what Dr. Whiting had said—if there is a normal fluid and no temperature, a radical operation is done and no evidence of visible necrosis is found, it would be better policy to let the labyrinth alone.

Referring to Dr. Kerrison's statement about the danger of a spinal puncture in an acute labyrinthitis, Dr. Smith said he thought that was more theoretical than practical, for in the case of acute labyrinthitis referred to in the paper, the patient started out with labyrinthian and meningeal symptoms, repeated lumbar puncture was done and though the count went to 8,000, the patient recovered. Two years ago, he had treated, at the Polyclinic, two cases of fractured skull with mastoiditis and secondary meningitis. The fractures ran across the mastoid to below the temporal ridge. The two cases occurred within six weeks of each other. The first patient was unconscious and had a stiff neck; spinal puncture showed a cloudy fluid with no bacteria. The next day he seemed a little better and another lumbar puncture was done. A secondary mastoiditis developed, he was taken to the operating room and a thorough mastoid operation was performed and he promptly died. Evidently the adhesions were broken up, death resulting. Within six weeks, the other case was brought in, unconscious and with a stiff neck, cloudy fluid and no bacteria. Here the puncture was repeated daily; each time the patient showed improvement. Then drainage was obtained by simply removing the cortex. Three months later, the patient was demonstrated in the clinic, had a convulsion, was found to have a large brain abscess and eventually died. The above cases both showed improvement following repeated punctures. Dr. Smith said that in his opinion, repeated lumbar punctures, instead of being detrimental, are a distinct aid, especially where there is a tendency towards localization of the process.

Dr. Smith said that he understood Dr. Kerrison to say that in case of accidental injury to the labyrinth during operation, he would do a labyrinth operation where labyrinthian symptoms developed, but had evidently misunderstood him and agreed that the case should be kept under observation and a labyrinth operation performed only where meningeal involvement indicated it.

DR. KOPETZKY said it is only by reporting these border line cases that definite conclusions can eventually be reached. Every one encounters these problems and they should be brought before the section and presented from the personal point of view.

Facial Paralysis and the Surgery of the Facial Nerve, with Lantern Slide Demonstration. Dr. K. W. Ney.

(*Published in full in this issue of* THE LARYNGOSCOP.)

DISCUSSION.

DR. BERNARD SACHS said he had been much interested in Dr. Ney's original contribution to this subject and the Doctor deserved much credit for the careful preparation and presentation of the subject. Probably all would agree with him in the condemnation of the former procedures that had been attempted for the cure of facial paralysis; the anastomoses

between the facial nerve and the accessory were not very successful. He, himself, had it tried on one or two cases, and abandoned it almost as soon at it was developed.

Dr. Sachs said he was a little in doubt about some of the points that Dr. Ney brought out, and probably in the hurried presentation of the subject he omitted to give all the information on one or two points, for instance, on what sort of cases should this operation be done? In what cases can it be expected to succeed? Dr. Sachs said he had always found it unfortunate for the facial nerve and perhaps still more unfortunate for the otologist, that the facial nerve was confined in this canal, and perhaps it was because it was so completely exposed that the otologist did the operation so successfully.

Dr. Sachs said he would also like Dr. Ney to state whether or not it would be applicable to the average case of Bell's palsy. Did he think the operation could be successfully applied in a large number of these cases? If he had understood correctly, Dr. Ney had expressed the opinion that the ordinary Bell's palsy was due to an involvement of the nerve in the outward fifth or sixth part of the passage of the canal. That was a surprise to him, and he would like very much to know upon what evidence that point rested, for he was not familiar with it. It would be very desirable to find a surgical cure for the large number of cases of Bell's palsy that are met with, and he was very curious to know to what extent one could look for improvement by surgical treatment of these cases. He had no desire to be critical, but felt slightly suspicious that the differentiation between the various strands in the peripheral nerve might not in every instance be so definite as Dr. Ney had pictured, though here again he was willing to yield to a more intimate knowledge of nerve structure.

Dr. Sachs said he was very glad Dr. Ney had laid some stress on the electrical examinations, for this was very frequently neglected in cases of ear disease where there was a suspicion that the nerve was involved. In a considerable number of cases in which it had been involved, whether associated with ear disease or not—the definite evidence of its involvement had been shown by the early results with the Faradic current, these tests being more important than the tests with the galvanic current or mechanical irritability. As long as any Faradic response is obtained, the hope of definite recovery may well be maintained. Once the Faradic response is lost, the course of the involvement of the nerve may be definitely stated to be very serious. The surgical propositions were entirely new and Dr. Sachs said he hoped Dr. Ney would not take it amiss that he had asked for elucidation on the points he had raised.

Dr. Israel Strauss said that Dr. Sachs had covered the points that the neurologists would like to bring forward, and he could not add much to the discussion . As Dr. Sachs had said, the first thing the neurologists wished to know was whether Dr. Ney intended this operation to be used in cases of the so-called idiopathic palsy, commonly known as Bell's Palsy, or in those which the otologists were so unfortunate as to cause at times. As all knew, a large proportion of the cases of Bell's palsy got well; a few did not. As to the pathology of Bell's palsy, very little is known, no pathologist has been able to see the facial nerve at any time when the pathology would be of any service in determining what the etiology was, so that it is a question whether it is advisable to go into the canal in that type of case and subject the patient to such an operation with the hope that there might be a constricting process present causing the disturbance. Dr. Ney was perfectly correct in supposing constriction in most of these cases, for his vast experience during the war in handling this nerve had shown how important it is to relieve any constriction of the nerve, no matter how slight; but it was questionable whether such an operation should be performed on a case of Bell's palsy, as there was great danger of injury to the middle ear by such a radical operation as the removal of all the bony parts which were described. Would it not endanger the ear function or subject it

to possible infection? That would be the first question the neurologist would put to the surgeon in a case of this kind. It was true that Bell's palsy was disfiguring, but it was an affliction that could be borne. One of the best neurologists in the profession was afflicted in this way, but it did not handicap him in his work. It was important to know exactly what were the dangers attending an operation of this kind.

In considering the cases of injury at the mastoid operation the proposition was a different one, for the otologist was in a measure prepared for such an operation and would be desirous of having such an injury repaired if possible. Dr. Strauss said that although he was not a surgeon, he had seen quite a little neural surgery, and as Dr. Ney described the operation and described it step by step it seemed comparatively simple; but the handling of a nerve the size of a lead pencil and in this locality was an extremely tedious operation and should be left in the hands of the neuro-surgeon. Very few even of those were competent to do work of this character. Dr. Ney, however, had opened up an opportunity for the relief of those unfortunate persons who had suffered from destruction or compression of this facial nerve, especially for those who following an operation have the tone of the muscle impaired. In some cases, there may be no function in the lower branch of the facial, but nearly a year later there is beginning function in the orbicularis. That shows that in that case the nerve is not completely destroyed; the nerve was not cut, but compressed, and although a year may have elapsed, experience in the war had shown that not merely a year, but two years may elapse between the time of compression and the removal of the compression, and the nerve may still have power to regenerate.

Dr. Whiting said that most of the phases of the proposition presented by Dr. Ney were entirely new to him. He knew something about nerve anastomosis that have been employed following accidents to the facial nerve and all the results that he had observed, after anastomosis, with the hypoglossal or spinal accessory, had been unsatisfactory; but had had no experience with the operation suggested, which was practically a decompression operation. The steps which Dr. Ney took in freeing the nerve from its canal appealed very strongly to him, for in the last three months he had himself exposed the nerve, completely, about thirty times. He had been doing this, not with any thought of attempting the operation, demonstrated by Dr. Ney, but from an entirely different standpoint. The procedure was not so extremely difficult, perhaps, as Dr. Sachs seemed to think. ,

Dr. Whiting said that he had been particularly impressed with the fact that Dr. Ney had also felt the necessity of removing the auditory plate in exposing the nerve from the stylo-mastoid foramen. That was the key to that step. As for exposing the nerve in the remainder of its course, Dr. Whiting said he had a set of very small chisels which accomplished this exposure adequately. It was not so difficult, as might be imagined, particularly in dealing with a nerve which had been paralyzed—when the operator would not experience the severe strain to which he would be subjected in exposing a living nerve with the attendant risk of seriously damaging the nerve sheath.

In reply to Dr. Strauss' question as to whether the nerve could be exposed from the stylo-mastoid foramen, particularly up to the geniculate ganglion without injury to hearing, would say that it could not be done without great impairment.

Dr. Friesner said that the cases in which such a procedure seemed feasible were two: first, those in which a fracture of the petrosa had occurred and in which the question of injury to the labyrinth would make little or no difference. From the otological point of view there would be a vast difference in the advisability of such a procedure, depending upon whether the nerve was injured in a simple or a radical mastoid operation. In a simple mastoid operation, the nerve would be injured in the vertical portion and there the procedure would be com-

paratively simple and quite feasible. In the radical operation, however, the nerve was usually injured in its tympanic course, frequently by the back of the curette. Any otologist who would have the temerity to perform a radical mastoid operation must know enough not to cut the nerve in two. In that regard Dr. Ney's explanation of the paralysis following the radical operation was perhaps correct, in that it was due to callous or cicatricial tissue in the nerve, not to an actual severance of the nerve itself.

Dr. Friesner said that in his own teaching of the anatomy of the temporal bone during the last twelve years, he had many times dissected out the facial nerve. There are temporal bones in which it is placed, not only below the external semicircular canal and between it and the oral window, but occasionally the external canal actually overhangs the aqueductus fallopii. Where the nerve takes a course of that kind, it is almost impossible to expose the tympanic part without injuring either the window or the external semi-circular canal. When it is injured in the simple mastoid operation, the exposure is simple, but the exposure of the tympanic part offers great technical difficulty that must endanger structures in close proximity where the injury would be serious or even vital.

Dr. Page said that he was much interested in the subject of Dr. Ney's paper and would like to ask a few questions, for recently he had had occasion to operate on a child where the nerve was severed below the tip; the doctor who operated said that the child had a celiulitis of the neck at the time, he operated on the mastoid and in trying to prick the cellulitis he severed the nerve at the tip, or in that region. That was done last May, and the child's mastoid had not healed. There was an injury to the membranous canal at the time of the operation. When the child came under his observation it had almost an atresia of the canal, with a foul discharge from a very small meatus, and on operating he found a necrosis of the bony canal; the antrum had not been opened and the mastoid had to be cleaned out. This was done, and the mastoid portion of the facial canal was found to be intact. There had been no injury in either the vertical or the tympanic portion. It was evident that there had been no invasion of that part. The injury took place at the tip, and there was considerable scar tissue and retraction in that region. What was Dr. Ney's opinion in regard to repair in that region if the nerve was cut—where all this inflammation had taken place and there were stellate scars? What hope was there of dissecting the nerve in that region and freeing it from the cicatrices that bound it down? And how long should one wait before operating or investigating? What tests would demonstrate that the nerve was or was not destroyed beyond repair?

Dr. Ney said that the danger of the operation, insofar as it might affect the hearing, depends entirely upon what portion of the nerve is involved. He considers the operation applicable in all types of facial paralysis where the lesion is located in the temporal bone. Dr. Ney said that he was very glad to hear those gentlemen who had found in their experimental work that the facial nerve could readily be exposed throughout its course in the temporal bone. There is no doubt but that it was a practical procedure; and even in cases of long standing there was a possibility of affecting a cure. In one case of 19 years standing there was return of function in the facial muscles following nerve suture.

In regard to the location of Bell's palsy as being within the vertical section of the canal, an examination of the literature on the subject indicated that apparently in most cases the lesion was located below the corda tympani branch. He had not met with any case in which the involvement of the nerve extended sufficiently high to cause paralysis of the stapedius, which leads him to believe that most lesions of this type are located in the vertical segment.

In regard to the anamalies of the facial canal he believed they were rather rare. Occasionally the lateral semi-circular canal may overhang

the facial canal to a degree which might make the removal of the nerve rather difficult. Such anamalies probably did not occur oftener than in 2 per cent of cases so that it would be a complication which would not often be encountered. He had operated a case in which the lateral sinus had a forward location—about 3 mm. behind posterior meatal wall —and that even under such conditions he had found it possible to get out the facial nerve.

The electrical examination of the facial muscles is very important. If the faradic irritability of the facial muscles is entirely lost the prognosis is grave; if it has been retained, the prognosis is usually favorable.

Dr. Ney said that the compression hypothesis seemed to be the only way in which many cases of facial paralyses could be accounted for. The facial nerve having particular anatomical characteristic; the nearest analogy of which is found in the musculospiral nerve which stands next to the facial nerve in frequency of lesions. Dr. Ney said that he believed any persisting case of facial paralysis should have the facial nerve explored and that his experience with these lesions and lesions of other nerves led him to believe that the prospect of restoring function in facial paralysis was very favorable.

Lesions of the facial nerve located below the stylo-mastoid foramen are handled as any other nerve injury. In such instances it is usually necessary to remove the mastoid tip to gain full access to the nerve. In this position it may be decompressed or sutured, depending upon the nature of the lesion.

(Since reading this article one facial nerve has been decompressed in which the facial paralysis followed surgical intervention to relieve suppuration below the mastoid in an infant 5 months old. The facial paralysis had persisted until the time of the operation, when the child was 6 years old. The facial nerve was compressed by scar and had not been divided at the time of the operation. After removal of scar—decompressing the nerve—the child had restoration of facial movements within one week.)

SECTION ON RHINOLOGY AND LARYNGOLOGY.

January 25, 1922.

Presentation of a Case Showing the Repair of the Right Nose with a Temporal Artery Forehead Flap, Before the Flap is Returned to the Forehead. Dr. J. Eastman Sheehan.

The object of presenting the case before the Section at this time was in order to demonstrate the intermediate steps in my reconstruction cases. Before, you have only seen the end results of the reconstructed work. So many of the men have asked me to present such a case; I have consented to abide by their request.

History: The patient was wounded at Verdun, October 6, 1918, receiving a severe wound to the head, face and an ugly wound of the whole right side of the nose. He was operated twice in France, twice at Cape May, N. J., and once at the Walter Reed Hospital, Washington, where he was discharged in October, 1919.

He entered the Public Health Hospital, No. 38, N. Y., upon my recommendation.

Examination: General, appearance, fairly well developed, somewhat underweight; does not appear acutely or chronically ill; head, eyes, pupils equal, regular, react to light and accommodation; lids, normal—extra ocular movements normal; face, scarred considerably. There are three small, irregular scars over left frontal region. A scar two inches long extending transversely across root of nose. On right side of face is a scar six inches long, extending along lower border of zygoma, and extending upward along base of nose. Another scar two and one-half inches long, parallel with ascending portion of later scar one-half inch below it. On left side of face there is a large irregular scar extending from middle of cheek to ala of nose, joining scar there. Another curved

scar extending along medial and inferior margins of orbit. Nose, deformed due to loss of bone and cartilage. Depression of bridge. Right ala shows loss of tissue. Scars irregular extending across bridge. Ears, negative; mouth, negative; chest, normal; lungs, negative; heart, negative; abdomen, negative; extremities negative; reflexes normal.

Operation: Under local anesthesia, using injections of ½% Novocain, the extensive scarring of the right side of nose and face was excised and the right ala reconstructed. A pattern was taken of the exact loss and so mapped out on the forehead. To carry the part of the patterned skin of the forehead to the nose was accomplished by using a temporal artery forehead flap, with the result as you see it tonight. The flap has nicely taken and I am sure the results will be all that could be desired. After I return the flap to the forehead, which I purpose to do, I also hope to further reconstruct the ala so as to match it with the other side. Later I hope to insert a cartilage transplant to lift the bridge of the nose.

DISCUSSION.

DR. MACKENTY said that the work demonstrated by Dr. Sheehan was so technical that not many were competent to discuss it. He had seen some of the Doctor's work before and had been very much impressed with his skill in handling these cases, and the Society was to be congratulated upon having a member who understood the work as he did.

Some Clinical Observations on the Correction of External Deformities of the Nose by the Intra-Nasal Route. With Lantern Demonstration. Dr. Hugh B. Blackwell.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HARMON SMITH congratulated Dr. Blackwell on the success of his work. He regretted, however, that Dr. Blackwell found it necessary to introduce a piece of rib or cartilage from the septum for the correction of minor nasal deformities. That in his experience the deformities resulting, particularly from a sub-mucous resection, could be overcome by the injection of a small amount of paraffin and that irrespective of the criticism brought against this method, that when there was sufficient looseness of skin, an underlying support with little or no scar tissue holding the tissues down, and with no history of syphilis, tuberculosis or diabetes, that the injection of paraffin was the simplest and most effective method for correcting these deformities and unattended by any evil result. He believed also that in the hundreds of cases that he had injected himself, and in the hundreds of others that had come under his observation, that if there was any predilection for paraffinoma that he would unquestionably have at least seen one case during his years of experience. He beleived that it is quite possible that these tumorous conditions could result from the employment of a paraffin melting at too high a temperature, or from a commercial paraffin that was not sufficiently refined. He further stated that he could not see any justification for resorting to a complicated procedure for the correction of a minor deformity when fifteen or twenty minutes was ample time for the correction by the injection of paraffin and was essentially an office proposition. He wished it understood, however, that he did not advocate the injection of paraffin for the major deformities where the nasal bones were separated or where it resulted from traumatism followed by considerable scar tissue, or where syphilis had undermined the nose and pulled it down by fibrous contraction within the lumen of the nares.

DR. HAYS expressed gratification that Dr. Blackwell had brought up this subject and hoped that Dr. Carter, who had done such excellent pioneer work along this line, would discuss the matter. Dr. Tieck, of this city, has also done admirable work of this kind; Dr. Hays said that, although he himself has done plastic work for many years, he did not know how simply it could be done until he saw Dr. Tieck's work. The rhinologists should do more of it, and not allow a patient to leave the

office with the statement that nothing can be done, only to drift later into the hands of the fakir.

These cases divide themselves into two classes: one composed of society women with minor deformities which they desire to have corrected, which can be done, although one is almost sure to get into trouble with malpractice suits later on. The other is composed of those physical and mental sufferers from nasal deformities who should have them corrected. It is surprising what can be done for a nose, and if a man uses the proper technique it does not make much difference what method he employs, so long as he has the proper artistic conception of what he has to do, and has a visualization of the exact result desired.

One of the most difficult things is to resect an old fractured nose, and some of these cannot be corrected by the methods suggested by Dr. Blackwell. Dr. Hays said he had a patient, belonging to the Princess Pat Regiment, who was hit over the nose with the butt of a rifle so that his nose was way to one side of the face. At first he attempted to correct this by loosening the parts with a chisel, but was unable to go far enough. He then gave the patient an anesthetic and tried to do what Dr. Blackwell suggested, refracture the parts with a hammer, but was a second time unsuccessful, although he hoped to try again and meet with a better result. These cases are very difficult. Dr. Iglauer has an electric saw for use in these cases. The most important part of Dr. Blackwell's paper was in demonstrating that any competent rhinologist is able to do this work as well as he does his intra-nasal surgery.

Dr. Carter thanked Dr. Hays for acknowledging his efforts in the field of nasal reconstruction, and said that he had devised the operation of combined bone and cartilage transplantation which Dr. Blackwell had described some fourteen or fifteen years ago, and had gone a great many cases since, a number of which had been shown before the Section. Dr. Carter said that it had always been his idea that the way to correct a nasal deformity is to use the patient's own tissue, so that when the deformity is corrected the continued growth will be along natural lines. The importance of this point had much to do with his introducing this operation, for the use of foreign bodies, which had been used prior to this time, had proved very unsatisfactory. In describing Dr. Carter's operation, Dr. Blackwell did not emphasize the fact that the bone should be brought into intimate contact with live periosteum-covered bone. The upper end of the transplant should be mortised into the frontal bone just above the naso-frontal suture; it should be slipped under the periosteum. The soft issues over the nose and in some intances over the cheeks should be well elevated so that they can be easily pulled towards the median line when the transplant is introduced. There will be a great deal of pressure on the transplant after the cicatricial tissue forms, and this together with the elasticity of the skin will tend to reproduce the deformity. In many instances the pressure is so great that it bends the transplant and in one case it fractured it. In order to obviate this, Dr. Carter modified his origial operation by introducing a bone or cartilage strut into the septum between the columellae to hold up the lower end of the dorsal transplant; this expedient has proved very satisfactory.

An important point which had not been mentioned is that one should over, rather than under-correct, in order to make allowance for cicatricial contraction and elasticity of the skin.

Referring to the deformities that sometimes result from the submucous operation, Dr. Carter said that he had seen a number of these and that he disagreed with Dr. Smith about using paraffin, though he recognized his mastery of this method. The Doctor had said that paraffin was not effective unless there was a firm foundation for it to rest upon; in these particular cases there is no foundation for it to rest upon. Even if there were no other reason the instances in which paraffinoma had been reported were so many that he would be afraid to use it himself. Recently he had read an article in which had been reported a number of cases of paraffinoma resulting from the use of liquid paraffin as a vehicle for the subcutaneous administration of drugs.

Referring to lateral deformities where force has been employed in making the correction, Dr. Carter said that he agreed with Dr. Hays when he said that many of these cases cannot be corrected as Dr. Blackwell indicated. The lateral deformity usually develops slowly, the skin accommodates itself to the deformity, one leg of the nasal arch is longer than the other; every influence tends to reproduce the deformity after it is corrected. Anyone can correct it by force, but no one can keep it there unless the bony and cartilanious structure of the nasal arch is properly corrected. Otherwise when the cast is taken off, inside of two months the nose will be back where it was before. The only way to prevent that is to take out a wedge-shaped portion of the long side of the arch. Dr. Carter said that he uses his chisel-forceps in liberating the nasal bones, and his modified Adams forceps in mobilizing the entire arch, then if the tissues are properly elevated and the entire nose put in a suitable splint a permanent correction may be effected.

Dr. Carter said that there is nothing so convincing to the lay mind as a well arranged gallery of "Befores" and "Afters." These "Before" and "After" photographic demonstrations are readily accepted as permanent results by the layman, by the general practitioner and by the surgeon who is inexperienced in nasal deformity work. These pictures are frequently taken shortly after operation and are misleading. The correction of a nasal deformity can never be judged a short time after the operation, for the ultimate controlling factors are cicatricial contraction and the elasticity of the skin. A perfect result immediately after the operation may be considered the opposite six months later.

Dr. Carter said that it was gratifying to note that appreciation for the importance of this work is being shown by the large number of men who have recently become interested in it.

DR. A. MICHAELIS inquired the percentage of infections that followed these intra-nasal methods, and said that some arguments had been brought against such methods and external methods advised in preference. It did not seem possible that any considerable number of operations of this kind could be done intra-nasally without causing some of these infections under the bridge of the nose. Even the submucous resections sometimes gave rise to abscesses.

DR. SHEEHAN said that the depressed and hump noses were as a rule quite easy to correct, but he quite agreed with Dr. Hays that the great problem lies in the depressed noses of syphilitic origin. Dr. Blackwell referred to these sort of cases, but he did not tell us what his modus operandi was, for their correction. I hope Dr. Blackwell will make this point clear.

The perichondrium should always be removed from the cartilage graft, otherwise, after due course it will curl up.

Referring to Dr. Corwin's inquiry about infections, Dr Sheehan said that he thought the use of intranasal routes was entirely wrong; it is unsurgical, and should be superceded by the external procedures. The reaction is tremendous and infection many times certain.

A new procedure, using an external pathway, which I have used for the past year, although I have not published it as yet, is as follows: The same incision is made for relieving depressions of the bridge, humps and deflections of the nose. An incision is made throughout the full length of the columella, extending from the tip of the nose down to the deep tissues of the philtrum. The septal membrane is now separated for a short distance. In the case of nasal deflections, after the loose tissue of the nose is freed, the membrane lining the nasal and nasal process of the maxilla is separated, the periosteum is then reflected from the outer side of the bone. The thickened, exostosed, positive side is removed by rongeuer forceps, chiseled away, or crushed inwards after chiseling. He quite agreed with one of the members, that the whole positive side should be removed. This was his own method of procedure in many cases. This new incision makes an excellent approach for the removal of hump and relieving depressions of the nasal bridge. There is very little scaring, reaction or infection. In cases of

marked depression of the bridge and where the tip has to be built up, the method gives the most satisfactory result of any procedure I have ever attempted.

As to the matter of paraffin. The results I have seen with the use of paraffin were generally bad. I have operated upon at least six cases where paraffin was used. It was a trying ordeal in some to remove all of it before I attempted to insert my bridge graft.

DR. LUIS P. BERNE said that he did not agree with Doctors Blackwell and Hays, in their opinions that the correction of a nasal hump or a deformity is a simple process.

The mere removal of a hump or angulation of a nose may be a simple matter, but the crux of genuine rhinoplasty lies not in the removal of a hump, but in the knowledge and appreciation of the conformation and correction of the lower and soft portion of the nose. Dr. Blackwell did not say a word about the correction of the lower or cartilaginous portion of the nose.

Dr. Blackwell showed slides, illustrating noses minus the hump, but not a word was said about the foreshortening which becomes necessary, as a rule, after such removal.

A hump should not be removed with bone-cutting forceps. Such manoeuvre is both crude and uncertain for instead of removing the nasal arch, the forceps may fracture the various bones which constitute the nasal arch.

Rhinoplasty, like plastic surgery, should convey besides ingenuity, the process of forethought the keen sense of patience combined with precision. Let us use the saw and let us know exactly where the nasal bones or frontal processes of the superior maxillae will sever.

The same is true of the attempt made by Dr. Blackwell of refracturing deflected noses with a mallet and a pair of forceps. Here likewise the saw should be employed and then alignment instituted.

Dr. Carter brought out a good point and one which I also employ in deflected noses of a marked degree; that is, to remove a wedge-shaped piece of bone from the concave side of the nose and merely saw through the convex side. This will prevent any possible overlapping.

I desire at this meeting to call attention to that type of nose where there is a thickening of the upper part of the upper lip and fronum. Here, as I have described in my recent article, I remove either the undue cellular thickening or the enlarged nasal spine or both. The nasal spine is cut away with saw and chisel. The excess cellular tissue is excised by carrying the knife underneath the skin of the upper lip, through the nares while a guilding finger is kept on the alveolar surface of the lip. The lip is now secured by passing a suture through the skin from one nares to the other and firmly tying the same.

DR. BLACKWELL wished to express his appreciation for the free and open nature of the discussion; he said he did not believe that Dr. Smith quite understood that the use of the rib graft was only recommended in the cases of very flat and sunken noses, eg.: saddle back deformities, etc. Owing to the great deformity present he did not believe that such cases could be readily corrected by means of paraffin injection. In the comparatively slight depressions where paraffin had been so successfully used by Dr. Smith, it was equally possible to secure a good reult by means of cartilage taken from the septum, the rib graft not being necessary in these cases.

DR. HAYS said that he had been unable to mobilize the nose by using a mallet; in some cases the author stated that it had been his own experience; in such instances it was necessary for him to first fracture the nasal processes of the superior maxillae by means of a strong pair of fracture forceps as described in his paper. This having been accomplished a light blow of the mallet on the lateral aspect of the nose was usually sufficient to loosen the attachment of the nasal to the frontal bone. As for removing a section from the long side of the nose so that it should remain straight, as recommended by Dr. Carter, Dr. Blackwell said he saw no reason why the bone should not be permitted to

override. He himself had never found it necessary to remove a wedge-shaped piece from the flat side in order to correct the condition, and in none of his cases had the deformity returned. He believed that the most frequent cause of the return of the deformity which had been noted by others was due to the fact that the nose had not been sufficiently mobilized at the time of the operation. He had never found a retention apparatus necessary to hold the nose straight during the process of healing. He had had two cases of infection, neither were hard to manage, both were drained intra-nasally with good ultimate results. Referring to the syphilitic cases of which Dr. Sheehan spoke, Dr. Blackwell said he would first try to secure a negative Wassermann by means of general treatment and only recommend the use of a graft where there was some underlying bony support present. In conclusion, he wished to emphasize the improvement in nasal breathing which followed the operations for the relief of lateral deformities and dropped tips and which justified some of the operations, not only for cosmetic, but for physiological reasons as well.

Case of Cut Throat. Frontal Lobe Abscess. Dr. Francis W. White.

W. S., 36. Two and a half years ago patient's throat was cut. Rushed to a hospital and tracheotomy tube was inserted, and the remainder of the cut area sutured. Tracheotomy tube remained in place three days. Patient remained in hospital three weeks.

He suffered more or less from respiratory embarrassment, which became suddenly worse about a week after his discharge from the hospital, patient falling to the sidewalk and being rushed to a nearby hospital, where a tracheotomy was performed under local anesthesia. He remained in the hospital ten weeks, after which time he applied for treatment at the Manhattan Eye, Ear and Throat Hospital.

There was muco-purulent discharge around the tracheotomy tube. He was put to bed, and the condition treated conservatively until the suppurative process ceased. Laryngotomy was performed, and a considerable amount of fibrous tissue in the form of a web, below the two cords, was removed, also granulation tissue in the larynx and trachea. Considerable portion of the cartilaginous rings from the tracheotomy opening upward had been replaced by new growth tissue. Thus, explaining the sucking in of the corresponding area when the patient tried to inspire with the trachea opening closed. Suturing of the thyroid cartilage was purposely omitted, hoping that a psuedo-arthrosis, false, or fibrous joint might be formed, thus increasing the transverse diameter of the larynx. As soon as the reaction of the operative procedure had subsided, gradual dialation of the larynx and trachea was instituted. First by means of ordinary laryngeal application. Later by means of intubation tubes, graduated sizes, and finally constant dialation by means of rubber tubing doubled upon itself and left in place twenty-four to forty-eight hours at a time. This method was successful, except that at the point on the posterior tracheal wall, directly opposite the curve of the tracheal tube, where there was a swelling or protuberance, which is found in nearly all cases of long duration tracheal tube breathing. This was overcome by performing a low tracheotomy, and simply using rubber tubing of greater length and bringing pressure to bear upon the above noted area.

J. G., 45. This patient swallowed a piece of lamb bone which stuck in his throat. He visited several clinics and when first seen by me there was a small pointed granulation in the right side of the esophagus below the cricoid.

He gave a history of night sweats and a history of diagnosis of both cancer and tuberculosis. I lost track of the patient for several weeks, but finally persuaded him to submit to operation, he having in the meantime, after a coughing spell, expectorated a piece of bone 1x¼ inches in size. An external incision was made at the point of pressure from the tip of a large urethral sound introduced through the patient's mouth and brought in contact with the small granulation mentioned previously. In this way a much smaller external incision was neces-

sary and dissection carried out directly to the abscess cavity which drained. Both internal and external openings closed rapidly.

Ida de G., 11. Applied at the Manhattan Eye, Ear and Throat Hospital in October, 1919, for treatment on account of swelling of the left upper eye-lid, headache and severe drowsiness.

Seven years previously she met with an accident, causing a compound comminuted fracture of the skull, since which time her mentality has been such that it was necessary to put her in an ungraded class in the public school. There was also a tendency to vomiting when riding in cars, and bright lights caused headaches.

Three days after admission, patient was operated upon by Dr. McCullagh, who reported this case. The left frontal sinus was opened and free pus and granulation tissue were found, and a frontal lobe abscess. Cultured pus from the sinus and drained abscess showed no growth after 120 hours.

The patient improved daily and convalescence was uninterrupted, despite the fact that she was very intractable and fought all attempts of dressing the wound. She was discharged from the hospital approximately four weeks from the time of admission.

A communication from the principal of her school, several months later, stated that her attendance had been very irregular and that she had not shown any improvement in her school work, but that her general behavior was very much better.

About a year later she was brought to the Manhattan Eye, Ear and Throat Hospital, presenting the following symptoms:

Fever, vomiting, swelling and pain over left eye, drowsiness and irritability when disturbed.

Physical examination: Somulent, very irritable when questioned, complained of headache and photo-phobia. Temperature 101.4. Marked rigidity of the neck, Kernig sign very marked in both legs, eye grounds negative. Within twenty-four hours temperature rose to 104.4.

Operation: Incision was made in the line of the scar of the previous operation and the former cavity was cleared of fibrous and granulation tissue, which showed very severe inflammatory reaction. A very small quantity of creamy pus was discovered. Exploratory punctures into the brain substance, in all directions, were negative.

Culture made, drainage provided and wet dressing applied. Temperature of the patient upon returning to the ward was 105.2. On the second day the temperature was 98.4, the next day a rise of temperature to 104.2, then to normal followed by a rise to 101.4, then reached 101 when there was a sudden rise to 105. From this time on, every third or fourth day, there was a rise of temperature, between 102 and 104, for nine consecutive times.

Despite this peculiar temperature range the morning following the operation the patient was much brighter, responded much more quickly to questions, and did not complain of pain. There was a gradual diminution of neck rigidity, and by the fifth or sixth day the neck symptoms and Kernig sign had disappeared.

With each of the sudden rises of temperature previously noted, the patient complained of severe lumbar pain, and upon two occasions it became necessary to administer an opiate.

At no time has the patient had chills or sweats. Repeated examinations of the blood for malarial elasmodia were negative.

White cells, 28,000 day of operation, and from that time on, between 8,000 and 12,000. Urinalysis, negative; spinal fluid, negative; blood culture, negative after 18 hours; culture from brain abscess, profuse growth of streptococcus capsulatouis; X-ray of spine, negative.

DISCUSSION.

DR. GLOGAU said that Dr. White was to be congratulated upon his good judgment in performing the external operation so early. He himself had in a recent paper before this Section tried to demonstrate the dangers of foreign bodies in the esophagus that may easily lead to mediastinitis and sepsis. If the incision is not made in the right direction,

it is very difficult to find the pus in these cases. Dr. White made the incision under an angle to the sterno-mastoid muscle. The method Dr. Glogau recently demonstrated went along the anterior margin of the muscle into the depth alongside the anterior margin of the vascular sheath. This is a logical procedure, as in any case of descending abscess the pus sooner or later will encroach upon the vascular sheath and from there burrow its way downward. In order to prevent the formation of mediastinitis during or after the operation, due to the downward oozing of pus, the collar mediastinum is sealed by iodoform packing simultaneously with the external drainage of the abscess. In case mediastinitis has been already established, the collar mediastinum is drained. V. von Hacker and Marschick have described the complications following foreign bodies within the esophagus and have urged the external operation.

Dr. J. M. McTiernan said that this was another opportunity to impress upon rhinologists the importance of following up sinus conditions complicating brain abscesses. Quite often the rhinologist is entirely lost when his case shows symptoms of becoming intracranial. During the past three years he had seen at autopsy four cases of frontal lobe abscesses wherein, despite the fact that a correct diagnosis had previously been made, at operation the abscess was not located. This he believed to be due to the failure of the operators in not correctly exploring the frontal area. He cited a case (Camp Meade Base Hospital) wherein a correct diagnosis of frontal abscess had been made, but a prominent specialist using only a small sub-Q-needle with which to explore, failed to find an abscess, which on autopsy performed within eighteen hours, contained about an ounce of pus.

The proper method is to explore the area with a special brain canula, as used at the Mayo Clinic, and in those cases where the abscess is quiescent, one would have good results, for it is not the tendency of a brain abscess to destroy brain tissue, rather to wall itself off, and as it develops to push the brain tissue aside.

Dr. White said he had not expected much discussion on the cases, as they had been presented and thoroughly discussed before, but wished the members of the Section to see the patients months or years after the various operative procedures.

Referring to the case of frontal lobe abscess, of which so many cases are seen, Dr. White said that what Dr. McTiernan had remarked about using a larger caliber of canula in exploring was very good. Probably the caliber used by rhinologists was rather small, but one was apprehensive of doing damage. In this instance he had used a medium sized canula and explored in all directions. The point that impressed him most in these cases was the violent reaction to the small amount of pus present. It seemed probable in this case that the scar tissue had contracted so severely that it produced an inordinate amount of pressure, due to the horizontal and vertical incisions, and the small amount of pus under such circumstances would probably give more symptoms than a larger amount of pus under slight pressure.

When the cut-throat case was first shown, there were one or two points emphasized: first, primary suture; second, tracheotomy, for the average surgeon has a tendency to slip in a tube and sew up the wound, or the case falls into the hands of an interne. In one instance a surgeon had a case of attempted suicide (such as this case was), and as the cut was rather high up, as would be suicides usually put their heads back in order to make a better incision, and cut into the air passage, a tracheotomy tube was put into the larynx, and the lower end of the tube set up an abscess and the man died. In these kind of cases the specialist should be called, if possible, and the case treated by him.

In the esophagotomy case the operation followed an abscess, and having seen it several times and knowing its points of exit internally, it was simply cut down upon. It happened that the abscess was posterior to the sheath.

Lingual Thyroid. Lantern Slides. John E. Welch, M.D.

The first case was seen in August, 1921, with a history that one morning she suddenly felt a sticking sensation in the throat and thought she had swallowed a needle or pin. Some of the family looked in the throat and found a tumor. The family physician said that it was probably an enlarged gland on the back of the tongue and gave her a mouth-wash, which she used for some days. She was then sent to a surgeon in Newark, who pronounced the case very malignant and advised operation. After that she saw Dr. Robinson, who said it was not a cancer, but a soft tumor, and after treating the case for three weeks, he sent her to Dr. John Sharpe, who referred her to Dr. Welch with a diagnosis of probable fibro-sarcoma Examination showed a tumor the size of a walnut., which was not at the time giving any symptoms It was dark, dusky red and angry looking. Palpation of the tongue tissue around the tumor showed that apparently the tissue was not involved, and the opinion was expressed that it was a muscle tumor. She was sent to the operating room, and under ether anesthesia the tumor was very easily shelled out. Two ligatures were used, and deep sutures stopped the hemorrhage. The patient made a good recovery and left the hospital on the sixth or seventh day, well.

DISCUSSION.

DR. H. E. SMYTH said that some years ago he had read a paper before the Triological Society on this subject. At that time he had been able to collect 67 cases, including one from Dr. Harris, one from Dr. Corwin, and three of his own, and had concluded that these tumors caused little inconvenience except from rapid growth, or considerable size. One of his cases had a marked tumor which caused no trouble until it was accidentally discovered, and another, the oldest of the series, never knew of its presence.

DR. BLACKWELL said that some years ago he had a case similar to that reported by Dr. Welch, in which the swelling came on very suddenly, and apparently the tumor consisted of colloid material. He operated and the patient recovered without any special symptoms. The tumor was as large as an English walnut.

DR. DWYER said that twelve years ago, within a month there were two cases at the Manhattan Hospital. One tumor was removed, a supposed muscle tumor, and on section it was found to be an aberrent thyroid. Later, the patient developed myxoedema. When the second patient came in with a thyroid tumor it was not removed. Later on, after the first patient had developed myxoedema, and it was found that it was a thyroid, the literature was consulted and reports of other cases were found.

DR. MACKENTY said that he had had two cases of this type of growth and had operated upon the first some two years ago. It was a large tumor, and fearing trouble he did a tracheotomy. Later the patient developed myxoedema. Last year he saw a case which looked like a thyroid, and on that diagnosis being proved it was let alone. The patient has been kept under observation. Unless the tumors give trouble by the size it is better to leave them alone. The tumor on which he operated was so large that it interfered with the patient's comfort.

DR. CORWIN said he had reported on a case in a boy of thirteen, in which the mass was 1½ inches in diameter and presented a very firm, smooth tumor-like body at the base of the tongue, interfering considerably with deglutition and other functions. He also had a smaller tumor in front of the neck, and the thyroid could not be palpated. The procedure was to cut out a narrow median section. The fibrous capsule was found thick and contained fluid. The escape of this fluid permitted much shrinkage and after healing the mass which remained gave little disturbance.

The point of the above remark is in the added lines (which you did not hear). The object was to show that removal of the entire mass is not always necessary to the relief of obstruction.

DR. UNGER asked whether Dr. Welch might be able to help him clear up a case he had. The patient came for treatment complaining of pain in swallowing, from which she had suffered for two weeks. Examination showed a minute drop of pus on the dorsum of the tongue, an inch from the epiglottis in the center line; a probe could be introduced into the substance of the tongue for ¾ of an inch. Would Dr. Welch consider that the location for the internal opening of a thyro-glossol cyst? That was the diagnosis made.

DR. LEDERMAN said the case brought up the subject of endocrinology. While this was too extensive a field to enter upon at the time, it suggested the query as to whether in a patient of this type would it not assist in reaching a diagnosis by finding the patient's basal metabolism before any operation was performed; might not the case be influenced by administering to the patient posterior pituitary substance and noting the effect upon the remaining thyroid gland tissue.

A New Procedure for the Relief of Abductor Paralysis of Larynx. Dr. John E. MacKenty.

For cases in which breathing is but slightly obstructed, but in which there are paroxysms of asphyxia.

A central incision exposes the wall of the trachea at a point underneath the thyroid bridge. The thyroid bridge is cut and retracted. A small oval window 1 c.m. long and .6 or .8 of 1 c.m. wide is removed from the tracheal wall, leaving the mucus membrane intact.

The mucus membrane is slit in the long axis of the trachea and turned up into the wound.

The skin of the neck is then brought down into the wound and carefully united to the mucosa of the trachea. Lateral skin incisions and undermining are required to relieve tension. The subcutaneous tissue is removed from the flap.

Tension sutures are placed so as to relieve all pull upon the mucous membrane skin union. A medium sized tracheal canula is inserted and the wound treated like an ordinary tracheotomy, every care being taken to prevent infection. Whe the wound is healed, the tracheal canula is replaced by a silver plug long enough to just enter the trachea. This is set in a cross-piece (for holding the tapes) similar to that used in a tracheal canula. If good skin and mucous membrane union are secured the opening contracts about one-half, leaving a hole about 5 c.m. in all diameters.

The silver plug is worn all the time and removed only when a asphyxia spell occurs.

This method of treatment does not interfere with speech as does the cord excision, nor does it irritate the trachea as does the tracheal canula.

For cases in which breathing is obstructed to a degree sufficient to cause discomfort and impairment of health.

The same procedure is followed as outlined above, excepting that the window resection of the trachea is larger, 1½ cm. long and 1 cm. wide. A large tracheal canula is worn in this opening until healing and contraction occur. Then the tube is entirely dispensed with. The reasonableness of this procedure came to me as a result of considerable experience in the trachea-skin union in laryngectomy. Many of my laryngectomized patients in whom primary union was secured between skin and tracheal mucous membrane do not wear a tracheal canula and are consequently relieved of much tracheal irritation.

New Method of Relieving Tension in Cleft Palate Flaps. Dr. John E. MacKenty.

I here present my method of holding the flaps in apposition in the operation for cleft palate. It has been employed for three years. I use a strip of very thin and flexible lead six to eight cm. wide and 20 cm. long. In this a double row of holes are punched, 1 cm. apart. A stab incision is made behind the posterior end of the alveolar process and well out near the reflection of the buchal surface. An artery clamp is passed through this opening, emerging in the nasopharynx. The tis-

sues are then stretched toward the median line. The lead ribbon is then passed from the mouth side through to the nasopharyngeal space, crossing this it is brought out on the mouth surface of the opposite side. The ends of the ribbon are crossed and tied at a point where it is deemed that sufficient tension is exerted. It is then tied and the redundant ends cut off.

If necessary a second ribbon is similarly placed in the hard palate region, the incisions being made anterior to the emergence of the palatine arteries. The posterior ribbon relieves all tension on the soft palate stitches, especially during the act of swallowing.

The ribbons are removed with the stitches on the tenth day. This procedure in my hands has added 20% to the cures.

Sensitivity. Dr. A. A. Eggston. By Invitation.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. W. C. SPAIN emphasized the fact brought out by Dr. Eggston; namely, that anaphylaxis is not a pathological entity, but that the manifestations of shock differ in different species of animals though constant for any one species. As shown by Dr. Eggston, the location of the susceptible tissues of the guinea pig are chiefly in the musculature of the bronchi and bronchioles. If an autopsy is done on a rabbit, however, that has just died of acute anaphylactic shock, the lungs will not be greatly distended as in the guinea pig, but in a state of collapse. Upon attempting to perfuse the pulmonary vascular system of such a rabbit killed by shock, it will be found that a very great pressure (90 mm.' is often required to force saline solution through, whereas in the normal rabbit, 10 mm. is ample. Manwaring has shown that if the liver of the sensitized dog is isolated from the circulation by an Eck fistula or any other form of collateral circulation shock cannot be produced.

Just as in the guinea pig, the muscles of the bronchial tree; as in the rabbit, the muscles of the pulmonary vascular system; and as in the dog, the liver tissues are respectively, the chief sites of the susceptible tissues just so i man we nd that the skin, subcutaneous tissues, mucosa, and submucosa are the main locations of susceptible tissues in those conditions of man known as Hypersensitiveness. In Hay-fever the tissues chiefly affected are the mucosa of the nose and the conjuctiva of the eye; in Asthma the mucosa and submucosa of the bronchi and bronchioles; in Urticaria, the kin; in Angioneurotic Edema, the ubcutaneous, and probably, the submucous tissues.

Dr. Spain objected to the term anaphylaxis being applied to these reactions in man. The antibody-antigen reaction has never been demonstrated in man conclusively and until this is done, it is reasonable to raise an objection. In man, it is known that heredity plays an important part. In a study of over 500 cases, Cooke and Van der Veer have shown that a great number—67 per cent—had a history of inheritance from both father and mother, in both of whom one or more of these different manifestations had occurred. In the guinea pig heredity plays no part, except that it does occur, as shown by Rosenau and Anderson, through a sensitized mother, but the sensitiveness of the offspring lasts but a few weeks. Then again in man, drugs that are definitely not antigenic, will bring about the hypersensitive condition such drugs being aspirin, quinine, mercury, arsenic.

Dr. Eggston stated the fact that many individuals gave a reaction to horse serum upon first injection, and explained it by saying that he considered in many that this might be due to many previous inhalations of horse dander, the patients thus becoming sensitized. In our experience, the reactions to horse serum and horse dander are quite distinct, and because a patient reacts to the protein of one by no means indicates that he will react to the protein of the other.

At the new York Hospital Clinic, in the treatment of Hay-fever, it has been found that the injection of the pollen to which there is the most susceptibility, carries with it a great deal of protection from the others; in other words, if the patient is susceptible to Timothy hay, June grass, etc., it is not necessary to give injections for all of these, for they are usually biologically related and the injection for the one to which the patient is most susceptible, will protect from the others.